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NEWS 33 Apr 21 Indexing from 1947 to 1956 being added to records in CA/CAPLUS
NEWS 34 Apr 21 New current-awareness alert (SDI) frequency in
WPIDS/WPINDEX/WPIX
NEWS 35 Apr 28 RDISCLOSURE now available on STN
NEWS 36 May 05 Pharmacokinetic information and systematic chemical names
added to PHAR
NEWS 37 May 15 MEDLINE file segment of TOXCENTER reloaded
NEWS 38 May 15 Supporter information for ENCOMPPAT and ENCOMPLIT updated
NEWS 39 May 16 CHEMREACT will be removed from STN
NEWS 40 May 19 Simultaneous left and right truncation added to WSCA
NEWS 41 May 19 RAPRA enhanced with new search field, simultaneous left and
right truncation

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This file contains CAS Registry Numbers for easy and accurate substance identification.

⇒ d 129:23341 all

ANSWER 1 CAPIUS COPYRIGHT 2003 ACS
AN 129:23341 CAPIUS
TI Subchronic physostigmine pretreatment in guinea pigs: effective against soman and without side effects
AU Philippens, Ingrid H. C. H. M.; Busker, Ruud W.; Wolthuis, Otto L.; Olivier, Berend; Bruijnzeel, Piet L. B.; Melchers, Bert P. C.
CS Research Group Pharmacology, TNO Prins Maurits Lab (TNO-PML), Rijswijk, 2280 AA, Neth.
SO Pharmacology, Biochemistry and Behavior (1998), 59(4), 1061-1067

CODEN: PBBHAU; ISSN: 0091-3057

PB Elsevier Science Inc.

DT Journal

LA English

CC 1-11 (Pharmacology)

Section cross-reference(s): 4

AB The behavioral and neurophysiol. effects of the subchronically administered cholinesterase-inhibitor physostigmine (PHY) (0.025 mg/kg/h) either with or without the muscarinic antagonist scopolamine (SCO) (0.018 mg/kg/h) were detd. in guinea pigs. In contrast to a single injection of PHY, subchronic application by osmotic minipumps of PHY, even without SCO, caused no behavioral or neurophysiol. side effects. Also, the efficacy of such a pretreatment in counteracting soman-induced lethality and apparent symptoms of intoxication were detd. After subchronically administered PHY or PHY + SCO, the treated animals were protected against a 3 x LD50 dose of soman.

ST subchronic physostigmine pretreatment soman; scopolamine physostigmine subchronic pretreatment soman

IT Brain

(EEG; subchronic physostigmine pretreatment, with or without scopolamine, in guinea pigs -- effective against soman and without side effects)

IT Reflex

(acoustic startle; subchronic physostigmine pretreatment, with or without scopolamine, in guinea pigs -- effective against soman and without side effects)

IT Detoxification

(biol.; subchronic physostigmine pretreatment, with or without scopolamine, in guinea pigs -- effective against soman and without side effects)

IT Behavior

(shuttlebox performance; subchronic physostigmine pretreatment, with or without scopolamine, in guinea pigs -- effective against soman and without side effects)

IT Drug interactions

(subchronic physostigmine pretreatment, with or without scopolamine, in guinea pigs -- effective against soman and without side effects)

IT Muscarinic receptors

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(subchronic physostigmine pretreatment, with or without scopolamine, in guinea pigs -- effective against soman and without side effects)

IT Nervous system

(visual, visual evoked response; subchronic physostigmine pretreatment, with or without scopolamine, in guinea pigs -- effective against soman and without side effects)

IT 96-64-0, Soman.

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)

(subchronic physostigmine pretreatment, with or without scopolamine, in guinea pigs -- effective against soman and without side effects)

IT 57-47-6, Physostigmine

RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(subchronic physostigmine pretreatment, with or without scopolamine, in guinea pigs -- effective against soman and without side effects)

IT 51-34-3, Scopolamine

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(subchronic physostigmine pretreatment, with or without scopolamine, in guinea pigs -- effective against soman and without side effects)

IT 9000-81-1, Acetylcholinesterase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(subchronic physostigmine pretreatment, with or without scopolamine, in
guinea pigs -- effective against soman and without side effects)
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
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=> d 109:185141 all

ANSWER 1 CAPLUS COPYRIGHT 2003 ACS
AN 1988:585141 CAPLUS
DN 109:185141
TI Effect of carboxylesterase inhibition on carbamate protection against
soman toxicity
AU Maxwell, Donald M.; Brecht, Karen M.; Lenz, David E.; O'Neill, Barbara L.
CS U. S. Army Med. Res. Inst. Chem. Def., Aberdeen Proving Ground, MD,
21010-5425, USA
SO Journal of Pharmacology and Experimental Therapeutics (1988), 246(3),
986-91
DT CODEN: JPETAB; ISSN: 0022-3565
LA Journal
LA English
CC 4-3 (Toxicology)
AB The ability of the carbamates pyridostigmine and physostigmine to protect
against the lethal effects of soman, an extremely toxic anticholinesterase

agent, was measured in rats, guinea pigs and rabbits. Pharmacol. equiv. doses of these carbamates that inhibited 70% of the blood acetylcholinesterase in each species were injected i.m. 25 min before s.c. injection with soman. Pretreatment with either carbamate, in combination with 17.4 mg/kg of atropine, produced protection against soman toxicity in all species. When protection was expressed as the ratio between the soman LD50 values in carbamate-protected animals and control animals, this protective ratio varied 3-fold between species (2.1-6.1 for pyridostigmine; 2.2-6.6 for physostigmine). When protection was expressed as the difference in the soman LD50 values between carbamate-protected animals and control animals, this protective difference was consistent among species (126 .mu.g/kg). Species variation in protective ratios was obsd. largely because the control LD50 values defining soman toxicity in unprotected animals varied among species (20 .mu.g/kg in rabbits, 28 .mu.g/kg in guinea pigs and 126 .mu.g/kg in rats). The species variation of the soman LD50 values in control animals was eliminated by pretreating animals with cresylbenzodioxaphosphorin oxide, which reduced the species variation in soman detoxification. The LD50 values for soman in cresylbenzodioxaphosphorin oxide-treated animals (9.8-15.6 .mu.g/kg) did not differ significantly between species. Similarly, protective ratios for carbamates against soman in cresylbenzodioxaphosphorin oxide-treated animals were also clustered in a narrow range (8.5-11.4 for pyridostigmine; 9.0-13.4 for physostigmine) that did not differ significantly, regardless of species or carbamate. These observations suggest that diverse mammalian species provide consistent ests. of the degree of protection that carbamates will provide against soman.

ST soman toxicity carbamate protection; pyridostigmine protection soman toxicity; physostigmine protection soman toxicity; carboxylestenase carbamate protection soman toxicity

IT Organ
(acetylcholinesterase and carboxylesterase of, carbamates protection against soman toxicity in relation to)

IT Abdominal diaphragm

Blood

Brain, composition

Liver, composition

Lung, composition
(acetylcholinesterase and carboxylesterase of, carbamates protection against soman toxicity in relation to)

IT 51-55-8, biological studies
RL: BIOL (Biological study)
(carbamates protection against soman toxicity in relation to)

IT 9000-81-1 9016-18-6
RL: BIOL (Biological study)
(of organ, carbamates protection against soman toxicity in relation to)

IT 57-47-6, Physostigmine 155-97-5
RL: BIOL (Biological study)
(soman toxicity protection by)

IT 96-64-0, Soman
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(toxicity of, carbamates protection against)

=> d 82:164856 all

ANSWER 1 CAPLUS COPYRIGHT 2003 ACS
AN 1975:164856 CAPLUS
DN 82:164856
TI Effect upon drug toxicity of surgical interference with hepatic or renal function
AU Selye, H.; Mecs, I.
CS Inst. Medecine Chir. Exp., Univ. Montreal, Montreal, QC, Can.

SO Acta Hepato-Gastroenterologica (1974), 21(3), 191-202; (4), 266-73
CODEN: AHGSBY; ISSN: 0300-970X
DT Journal
LA English
CC 1-5 (Pharmacodynamics)
Section cross-reference(s): 2, 4, 13
GI For diagram(s), see printed CA Issue.
AB The effect of choledochus ligature, partial hepatectomy, partial nephrectomy, and the steroids, pregnenolone-16.alpha.-carbonitrile (PCN) [1434-54-4] and triamcinolone [124-94-7] on the toxicity of 175 drugs was detd. in rats. For example, the toxicity of glutethimide (I) [77-21-4] was inhibited by PCN and triamcinolone and increased by choledochus ligature, partial hepatectomy, and to a lesser extent, partial nephrectomy, whereas indomethacin [53-86-1] was detoxified by choledochus ligature and PCN but was unaffected by the other treatments. The toxicity of 77 compds. was decreased by PCN, but was potentiated by partial hepatectomy in only 53 of them. Triamcinolone inhibited the toxicity of 33 compds.
ST drug toxicity liver kidney surgery; triamcinolone drug toxicity; pregnenolonecarbonitrile drug toxicity
IT Detoxication
(of pharmaceuticals)
IT Kidney, metabolism
Liver, metabolism
(pharmaceutical detoxication by)
IT 124-94-7 1434-54-4
RL: BIOL (Biological study)
(pharmaceuticals detoxication response to)
IT 50-09-9 50-12-4 50-29-3, biological studies 51-12-7 51-28-5,
biological studies 51-42-3 51-52-5 51-56-9 52-31-3 52-86-8
53-21-4 53-86-1 54-11-5 54-21-7 54-64-8 54-95-5 55-65-2
55-86-7 55-91-4 56-34-8 56-38-2 56-81-5, biological studies
56-89-3, biological studies 57-06-7 57-30-7 57-44-3 57-53-4
57-83-0, biological studies 57-94-3 58-15-1 58-25-3 58-73-1
59-41-6 59-47-2 59-52-9 60-19-5 61-80-3 62-55-5 62-74-8
62-75-9 64-17-5, biological studies 64-31-3 64-47-1 64-86-8
66-76-2 66-81-9 67-21-0 67-66-3, biological studies 67-68-5,
biological studies 67-96-9 71-27-2 71-63-6 71-73-8 71-78-3
73-24-5, biological studies 75-15-0, biological studies 75-80-9
76-44-8 77-21-4 78-34-2 78-44-4 79-06-1, biological studies
81-23-2 81-25-4 81-81-2 82-58-6 83-44-3 83-67-0 86-50-0
87-66-1 89-82-7 94-59-7 95-25-0 97-44-9 98-50-0 100-17-4
100-61-8 103-72-0 103-84-4 103-90-2 107-07-3, biological studies
107-12-0 107-13-1, biological studies 107-21-1, biological studies
108-86-1 109-75-1 110-89-4, biological studies 114-49-8 118-74-1
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125-64-4 125-84-8 125-85-9 126-07-8 127-48-0 127-85-5 128-37-0
129-46-4 131-73-7 132-60-5 134-72-5 137-58-6 145-41-5 145-42-6
146-56-5 151-67-7 152-16-9 154-42-7 156-57-0 288-13-1 298-46-4
299-78-5 300-62-9 300-68-5 302-95-4 306-07-0 315-30-0 316-42-7
318-98-9 361-09-1 434-13-9 466-06-8 492-18-2 496-72-0 513-10-0
530-78-9 551-06-4 553-69-5 554-13-2 563-12-2 584-84-9 593-74-8
630-93-3 830-89-7 863-57-0 1095-90-5 1229-29-4 1303-28-2
1401-55-4 1421-86-9 1553-60-2 1639-60-7 1772-03-8 1867-66-9
2104-64-5 2181-04-6 3238-60-6 3820-67-5 4044-65-9 5341-61-7
5907-38-0 7447-39-4, biological studies 7487-94-7 7601-89-0
7723-14-0, biological studies 7785-87-7 7790-86-5 9011-04-5
10025-82-8 10099-58-8 10108-64-2 10138-52-0 13410-01-0
15256-58-3 15500-66-0 15571-91-2 15687-27-1 18911-13-2
39377-61-2 55347-53-0
RL: PRP (Properties)
(toxicity of, kidney and liver and steroids effect on)

=> d 66:17758 all

ANSWER 1 CAPIUS COPYRIGHT 2003 ACS

AN 1967:17758 CAPIUS

DN 66:17758

TI Protective effect of aldrin against the toxicity of organophosphate anticholinesterases

AU Triolo, Anthony J.; Coon, Julius M.

CS Jefferson Med. Coll., Philadelphia, PA, USA

SO Journal of Pharmacology and Experimental Therapeutics (1966), 154(3), 613-23

CODEN: JPETAB; ISSN: 0022-3565

DT Journal

LA German/French

CC 14 (Toxicology)

AB A single oral dose of 16 mg. of aldrin/kg. protected mice 4 days later against parathion, para-oxon, tetraethyl pyrophosphate, diisopropyl fluorophosphate, O-ethyl O-(p-nitrophenyl) phosphorothioate, Guthion, tri-o-tolyl phosphate, and physostigmine, but not against octamethylpyrophosphoramido (OMPA) or neostigmine. One hour after aldrin, the toxicity of parathion was increased, whereas, from 16 hrs. to 12 days after aldrin, animals were significantly protected. This effect of aldrin reached its max. in apprx.4 days, and 1 mg./kg. provided significant protection. Two days after aldrin, A-esterase, which detoxifies para-oxon, increased 38% in the liver but decreased 50% in the plasma, and plasma B-esterase, which is inhibited by para-oxon, was increased 24%. Aldrin had no effect on the inhibitory action of para-oxon on plasma cholinesterase, but it reduced this action of para-oxon in the brain. This is in accord with the finding that aldrin failed to protect against OMPA or neostigmine, which differ from the other anticholinesterases tested in being poor in vivo inhibitors of brain cholinesterase. Ethionine abolished the protective effect of aldrin against the toxicity and brain cholinesterase-inhibiting action of para-oxon and prevented the aldrin-induced increases in plasma B-esterase and liver A-esterase. Ethionine, alone, however, increased the mortalities after parathion and para-oxon. Though the increases in A- and B-esterases would be expected to decrease the toxicities of parathion and para-oxon, other factors possibly involving the central nervous system may play a role in the protective effect of aldrin against organophosphate poisoning.

ST ORGANOPHOSPHATES ALDRIN; ANTICHOLINESTERASE ALDRIN; ALDRIN PESTICIDES; PESTICIDES ALDRIN; PESTICIDES ALDRIN; ALDRIN PESTICIDES; ANTICHOLINESTERASE ALDRIN; ORGANOPHOSPHATES ALDRIN

IT Brain, composition

(cholinesterase inhibition by organophosphate in, ethionine inhibition of aldrin protection of)

IT Poisoning

(organophosphate, aldrin protection against)

IT 55-17-4

RL: BIOL (Biological study)

(aldrin protective action against p-oxon anticholinesterase action inhibition by)

IT 9013-79-0, Esterases

(in blood, brain and liver in organophosphate poisoning, aldrin effect on)

IT 9001-08-5, Esterases, choline

(inhibition of, by organophosphate in brain, aldrin protection of, ethionine antagonism to)

IT 309-00-2

RL: PROC (Process)

(organophosphate poisoning-protective action of)

IT 55-91-4 56-38-2 57-47-6 78-30-8 86-50-0 107-49-3 311-45-5

15576-30-4

RL: BIOL (Biological study)
(poisoning by, aldrin protection against)

=> d 103:66085 all

ANSWER 1 CAPIUS COPYRIGHT 2003 ACS

AN 1985:466085 CAPIUS

DN 103:66085

TI Interethnic differences of human serum paraoxonase activity-relevance for the detoxification of organophosphorous compounds

AU Geldmacher-Von Mallinckrodt, M.; Diepgen, T. L.; Enders, P. W.

CS Inst. Rechtsmed., Univ. Erlangen-Nuernberg, Erlangen, D-8520, Fed. Rep. Ger.

SO Archives Belges de Medecine Sociale, Hygiene, Medecine du Travail et Medecine Legale (1984), Suppl.(Proc.-World Congr. "New Compd. Biol. Chem. Warf.: Toxicol. Eval., 1st, 1984), 243-51

CODEN: ABMHAM; ISSN: 0003-9578

DT Journal; General Review

LA English

CC 4-0 (Toxicology)

AB A review with 19 refs. on interethnic differences of human serum paraoxonase [117698-12-1] activity and its relevance for the detoxication of organophosphorus compds., i.e., paraoxon [311-45-5].

ST review serum paraoxonase human genetics; detoxication organophosphate serum paraoxonase review

IT Detoxication

(of organophosphorus compds., interethnic differences of human blood serum paraoxonase in relation to)

IT Genetics

(paraoxonase of human blood serum in relation to)

IT Blood serum

(paraoxonase of, of humans, interethnic differences of, detoxication of organophosphorus compds. in relation to)

IT 311-45-5 7723-14-0D, org. compds.

RL: BIOL (Biological study)

(detoxication of, interethnic differences of human blood serum paraoxonase in relation to)

IT 117698-12-1

RL: BIOL (Biological study)

(of blood serum, of humans, interethnic differences of, detoxication of organophosphorus compds. in relation to)

=> d 102:161862 all

ANSWER 1 CAPIUS COPYRIGHT 2003 ACS

AN 1985:161862 CAPIUS

DN 102:161862

TI Metabolic activation of phosphorothioate pesticides: role of the liver

AU Sultatos, Lester G.; Minor, Lerna D.; Murphy, Sheldon D.

CS Med. Cent., Louisiana State Univ., New Orleans, LA, USA

SO Journal of Pharmacology and Experimental Therapeutics (1985), 232(3), 624-8

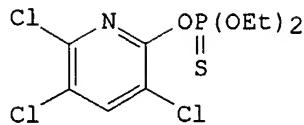
CODEN: JPETAB; ISSN: 0022-3565

DT Journal

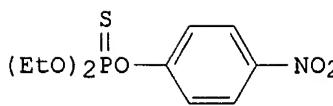
LA English

CC 4-4 (Toxicology)

GI



I



II

AB Mouse liver perfusion studies *in situ* revealed that the cholinesterase inhibitor chlorpyrifos oxon [5598-15-2] produced by the liver from the phosphorothioate pesticide chlorpyrifos (I) [2921-88-2] was quickly detoxified within the liver, thereby preventing its exit from the liver in the effluent. In contrast, when the pesticide parathion (II) [56-38-2] was perfused as a substrate, a substantial amt. of the toxic metabolite paraoxon [311-45-5] was found in exiting perfusate. Pesticide concns. (5-15 .mu.M) used in the perfusion studies *in situ* were similar to their hepatic portal blood concns. *in vivo* (2.32-12.95 .mu.M) after i.p. administration of lethal or near LDs. Moreover, the half-life for elimination of paraoxon by mouse blood *in vitro* was 8.6 min, a rate sufficiently low to allow passage of paraoxon to extrahepatic target tissues from liver *in vivo*. Apparently, in the mouse, the acute toxicity of chlorpyrifos is mediated by extrahepatic prodn. of oxon, whereas parathion is likely mediated by hepatic and extrahepatic activation.

ST liver chlorpyrifos parathion metab

IT Liver, metabolism
(chlorpyrifos and parathion metabolic activation in, perfusion in relation to)

IT Blood
(chlorpyrifos and parathion of, after administration, liver in relation to)

IT 311-45-5 5598-15-2
RL: FORM (Formation, nonpreparative)
(formation of, by liver, perfusion in relation to)

IT 56-38-2 2921-88-2
RL: BIOL (Biological study)
(metabolic activation of, liver perfusion in relation to)

=> d 101:165142 all

ANSWER 1 CAPLUS COPYRIGHT 2003 ACS

AN 1984:565142 CAPLUS

DN 101:165142

TI Paraoxonase and paraoxon detoxification

AU Butler, Edward Grant

CS Univ. Michigan, Ann Arbor, MI, USA

SO (1984) 111 pp. Avail.: Univ. Microfilms Int., Order No. DA8412112
From: Diss. Abstr. Int. B 1984, 45(2), 522-3

DT Dissertation

LA English

CC 4-4 (Toxicology)

AB Unavailable

ST paraoxon detoxication paraoxonase; paraoxonase paraoxon detoxication

IT 311-45-5
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(metab. of, in human and lab. animals, paraoxonase in relation to)

IT 117698-12-1
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(paraoxon metab. in human and lab. animals in relation to)

=> d 98:66771 all

ANSWER 1 CAPIUS COPYRIGHT 2003 ACS

AN 1983:66771 CAPIUS

DN 98:66771

TI Enzymic detoxication of organophosphorus insecticides and nerve gases in primates

AU Losch, H.; Losch, K.; Haselmeyer, K. H.; Chemnitius, J. M.; Zech, R.

CS Zent. Biochem., Georg-August-Univ., Goettingen, 3400, Fed. Rep. Ger.

SO Arzneimittel-Forschung (1982), 32(12), 1523-9

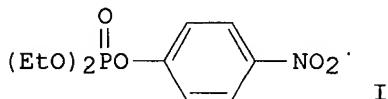
CODEN: ARZNAD; ISSN: 0004-4172

DT Journal

LA German

CC 4-4 (Toxicology)

GI



AB The detoxication of organophosphorus compds. by phosphorylphosphatases was studied in primates. Taking into account the distribution of paraoxonase and DFPase (EC 3.8.2.1) [9032-18-2] in different tissues of the monkey (Macaca mulatta), the total detoxicating capacity for paraoxon (I) [311-45-5] and diiso-Pr phosphorofluoridate (DFP) [55-91-4] was detd. acetylcholinesterase (EC 3.1.1.7) (AChE) [9000-81-1] of human brain was inhibited in vitro by I and DFP. By using the rate consts. of AChE inhibition and synthesis, the concns. of organophosphorus inhibitors were calcd., which would reduce the steady-state AChE activity to 20% of normal. This acute ineffective concn. is 7.6 .times. 10-8 g/kg for DFP and 2.3 .times. 10-8 g/kg for I. From substrate kinetics of the phosphorylphosphatases, the time course of I and DFP detoxication in primates could be calcd. The time needed by phosphorylphosphatases to reduce a certain dose of an organophosphorus compd. to the acute ineffective concn. is referred to as effective detoxication time. The effective detoxication time was detd. for different concns. of I and DFP and compared with the time needed by the organophosphate concns. to inhibit AChE activity to 12.5% of normal. The significance of in vitro data for the evaluation of dose limits of organophosphate toxicity in vivo is discussed.

ST paraoxon enzyme detoxification; phosphorofluoridate enzyme detoxification; enzyme detoxification org. phosphate

IT Brain

Kidney

Liver

Lung

Muscle

Organ

Spleen

(diisopropyl phosphorofluoridate and paraoxon detoxification by, in humans)

IT Kinetics, enzymic

(of acetylcholinesterase and diisopropyl fluorophosphatase and paraoxonase detoxification of org. phosphorus compds.)

IT Blood serum

(paraoxon detoxification by, in humans)

IT 55-91-4 311-45-5

IT RL: BIOL (Biological study)
(detoxification of, kinetics of)
IT 9000-81-1
RL: BIOL (Biological study)
(diisopropyl phosphorofluoridate and paraoxon inhibition of,
detoxification kinetics in relation to)
IT 9032-18-2
RL: PROC (Process)
(diisopropyl phosphorofluoridate inhibition of, detoxification kinetics
in relation to)
IT 117698-12-1
RL: PROC (Process)
(paraoxon inhibition of, detoxification kinetics in relation to)

=> d 95:215979 all

ANSWER 1 CAPIUS COPYRIGHT 2003 ACS
AN 1981:615979 CAPIUS
DN 95:215979
TI Biological effect of organophosphorus pesticides at low concentration. I.
The detoxication of fenitrooxon at low concentration by mouse liver
preparation
AU Kawamura, Youko; Takeda, Mitsuharu; Uchiyama, Mitsuru
CS Natl. Inst. Hyg. Sci., Tokyo, Japan
SO Eisei Kagaku (1981), 27(4), 252-6
CODEN: ESKGA2; ISSN: 0013-273X
DT Journal
LA Japanese
CC 4-3 (Toxicology)
AB Fenitrooxon (FO) [2255-17-6] (.apprx.10-6M) reacted with mouse liver
homogenate and disappeared immediately. This occurred mostly in the sol.
fraction of mouse liver, and was dependent on glutathione (GSH)
[70-18-8], and the only metabolite detected was desmethyl-FO [950-35-6].
On the other hand, since hydrolysis by arylesterase (AEase) [9032-73-9]
did not occur, 4-nitro-m-cresol was not detected. Apparently, at such low
concn., FO is detoxified solely through desmethylation reaction catalyzed
by GSH S-transferase (GTase) [50812-37-8]. The Km and Vmax values of
both GTase and AEase for FO are also consistent with this reaction
mechanism, i.e., detoxication of FO at higher concn. may be attributable
to both GTase and AEase, but at low concn. only GTase will be responsible.
ST fenitrooxon detoxification liver enzyme
IT Liver, metabolism
(fenitrooxon detoxification by, enzymes in relation to)
IT Michaelis constant
(of arylesterase and GSH transferase, of liver, fenitrooxon
detoxification in relation to)
IT 2255-17-6
RL: BIOL (Biological study)
(detoxification of, by liver, enzymes in relation to)
IT 950-35-6
RL: FORM (Formation, nonpreparative)
(formation of, in liver, mechanism of)
IT 70-18-8, biological studies
RL: BIOL (Biological study)
(liver detoxification of fenitrooxon in relation to)
IT 9032-73-9 50812-37-8
RL: BIOL (Biological study)
(of liver, fenitrooxon detoxification in relation to)

=> d 89:210014 all

ANSWER 1 CAPLUS COPYRIGHT 2003 ACS

AN 1978:610014 CAPLUS

DN 89:210014

TI Effects of naturally occurring food plant components on insecticide degradation in rats

AU Fuhrmann, Tom W.; Lichtenstein, E. Paul; Stratman, Fredrick W.

CS Inst. Enzyme Res., Univ. Wisconsin, Madison, WI, USA

SO Journal of Agricultural and Food Chemistry (1978), 26(5), 1068-75

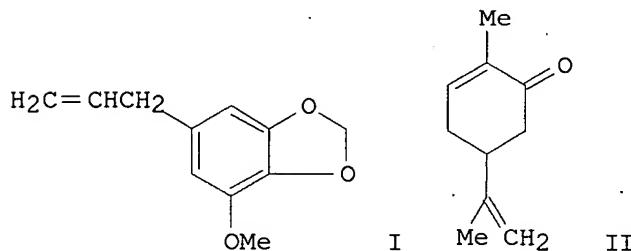
CODEN: JAFCAU; ISSN: 0021-8561

DT Journal

LA English

CC 4-4 (Toxicology)

GI



AB The effects of the naturally occurring, insecticidal, food plant components myristicin (I) [607-91-0] and d-carvone (II) [2244-16-8] on insecticide degrdn. by subcellular fractions of rat livers or by intact liver cells (hepatocytes) were evaluated. The naturally occurring compds. were incorporated into rat diets to det. their in vivo effects on insecticide degrdn. by subcellular fractions or hepatocytes. To det. their in vitro effects I and II were added simultaneously with the insecticides to subcellular fractions or hepatocytes. Insecticides studied were 14C-labeled parathion [56-38-2], paraoxon [311-45-5], and fonofos [944-22-9]. Results indicated that both I and II interacted with rat liver components to either increase insecticide degrdn. to detoxified metabolites or to block degrdn. as measured by an increased stability of the parent insecticide. Effects varied depending on the particular natural compd., the route of administration (in vivo or in vitro), and the particular liver cell fraction. The effects of feeding I and II were in most cases different from effects obsd. after their simultaneous in vitro addn. with the insecticides. The effects obsd. with these naturally occurring compds. in the living organism were not necessarily the same as those obsd. after their addn. to subcellular liver fractions. Hepatocytes were found to be a useful alternative technique for investigating insecticide degrdn.

ST insecticide metab liver carvone myristicin

IT Insecticides

(phosphorous-contg., metab. of, by liver, carvone and myristicin effect on)

IT Liver, metabolism

(phosphorus-contg. insecticides metab. by, carvone and myristicin effect on)

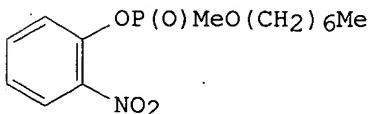
IT 607-91-0 2244-16-8

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(insecticide metab. by liver response to)

IT 56-38-2 311-45-5 944-22-9
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(metab. of, by liver, carvone and myristicin effect on)

=> d 88:184105 all

ANSWER 1 CAPIUS COPYRIGHT 2003 ACS
AN 1978:184105 CAPIUS
DN 88:184105
TI Detoxification of nitrophenyl phosphate and nitrophenyl phosphonates in
tissue homogenates of white rats
AU Galebskaya, L. V.
CS I Leningr. Med. Inst., Leningrad, USSR
SO Neirogumoral'n. Endokr. Regul. Funkts. (1975), 28-9. Editor(s): Denisova,
G. A.; Maslennikov, I. V.; Smirnova, N. N. Publisher: Pervyi Leningr. Med.
Inst. im. I. P. Pavlova, Leningrad, USSR.
CODEN: 37TFAW
DT Conference
LA Russian
CC 4-3 (Toxicology)
GI



I

AB 0-Heptyl-0-o-nitrophenyl methylphosphonate (I), [62704-83-0] its 0-pentyl
analog, [59223-32-4] and 0-heptyl 0-isopentyl 0-o-nitrophenyl phosphate
[59223-33-5] were hydrolyzed by rat liver homogenates at the P-O-aryl
linkage. The rate of overall hydrolysis for 16 times 10⁻⁴ M I plus 16
times 10⁻⁴ M paraoxon [311-45-5] was less than the total of the
hydrolysis rates for the 2 compds. and approximated the rate for I. The
heptyl radical apparently facilitates the steric fit between the
organophosphorus linkage and the enzyme active center.
ST nitrophenyl phosphate phosphonate detoxication liver
IT Liver, metabolism
(detoxication by, of nitrophenyl phosphates and phosphonates)
IT Detoxication
(of nitrophenyl phosphates and phosphonates, by liver)
IT 311-45-5 59223-32-4 59223-33-5 62704-83-0
RL: PROC (Process)
(detoxication of, by liver)

=> d 107:229142 all

ANSWER 1 CAPIUS COPYRIGHT 2003 ACS
AN 1987:629142 CAPIUS
DN 107:229142
TI Stimulation of defenses of biological systems using toxic substances
IN Berdal, Pascal
PA Fr.
SO Fr. Demande, 25 pp.
CODEN: FRXXBL
DT Patent
LA French

IC ICM A61K045-05
CC 1-4 (Pharmacology)
Section cross-reference(s): 63

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	FR 2584294	A1	19870109	FR 1985-10403	19850708
	FR 2584294	B1	19920221		
PRAI	FR 1985-10403		19850708		
AB	The defenses of biol. systems are augmented by administration of at least two substances chosen among: immunodepressants, immunotoxins, cytotoxins, cytostatics, and/or immunomodulators (no data). A synergistic effect occurs as these toxic substances stimulate the biol. system.				
ST	stimulation biol system defense toxic substance				
IT	Light (-like activity, stimulation of defenses of biol. systems using)				
IT	Inflammation (agents for stimulation of, stimulation of defenses of biol. systems using)				
IT	Catharanthus roseus (alkaloids from, stimulation of defenses of biol. systems using)				
IT	Toxins RL: BIOL (Biological study) (bacterial, stimulation of defenses of biol. systems using)				
IT	Enterobacteriaceae Escherichia coli Leptospira Proteus (bacterium) Salmonella Shigella (endotoxins from, stimulation of biol. systems using)				
IT	Tubulins RL: BIOL (Biological study) (inhibitors and antagonists of polymn. of, stimulation of defenses of biol. systems using)				
IT	Deoxyribonucleic acids RL: BIOL (Biological study) (inhibitors of synthesis of, stimulation of defenses of biol. systems using)				
IT	Peroxidation (of lipids, stimulation of defenses of biol. systems using product of decompn. of)				
IT	Lipids, biological studies RL: BIOL (Biological study) (peroxidn. of, stimulation of defenses of biol. systems using products from decompn. of)				
IT	Mitogens (pokeweed, stimulation of defenses of biol. systems using)				
IT	Corynebacterium Cytotoxic agents Hydroxyl group Immunosuppressants Inflammation inhibitors Microorganism Neoplasm, composition Neoplasm inhibitors Peroxisome Radiomimetics Venoms Aflatoxins Agglutinins and Lectins Aldehydes, biological studies				

Alkanes, biological studies
Antibodies
Leukotrienes
Mycotoxins
Prostaglandins
Radicals, biological studies
Ricins
Thromboxanes
Toxins
RL: BIOL (Biological study)
(stimulation of defenses of biol. systems using)

IT Lipopolysaccharides
RL: BIOL (Biological study)
(stimulation of defenses of biol. systems using bacterial)

IT Antibodies
RL: BIOL (Biological study)
(auto-, stimulation of defenses of biol. systems using)

IT Agglutinins and Lectins
RL: BIOL (Biological study)
(concanavalins, stimulation of defenses of biol. systems using)

IT Toxins
RL: BIOL (Biological study)
(endo-, bacterial, stimulation of defenses of biol. systems using)

IT Toxins
RL: BIOL (Biological study)
(immuno-, stimulation of defenses of biol. systems using)

IT Peroxides, biological studies
RL: BIOL (Biological study)
(lipid, stimulation of defenses of biol. systems using)

IT Antibodies
RL: BIOL (Biological study)
(monoclonal, stimulation of defenses of biol. systems using)

IT Lipids, biological studies
RL: BIOL (Biological study)
(peroxy, stimulation of defenses of biol. systems using)

IT Agglutinins and Lectins
RL: BIOL (Biological study)
(phytohemagglutinins, stimulation of defenses of biol. systems using)

IT 120-73-0D, Purine, biol. derivs. 289-95-2D, Pyrimidine, biol. derivs.
RL: BIOL (Biological study)
(inhibitors of synthesis of, stimulation of defenses of biol. systems using)

IT 56-85-9, Glutamine, biological studies
RL: BIOL (Biological study)
(inhibitors or antagonists of, stimulation of defenses of biol. systems using)

IT 149-29-1 9003-99-0, Peroxidase 9013-93-8, Lecithinase 9035-82-9
RL: BIOL (Biological study)
(simulation of defenses of biol. systems using)

IT 59-30-3D, Folic acid, analogs
RL: BIOL (Biological study)
(stimulation of defenses of biol systems using)

IT 50-18-0, Cyclophosphamide 50-44-2, 6-Mercaptopurine 51-18-3,
Triethylene melamine 51-21-8, 5-Fluorouracil 52-24-4, Triethylene
thiophosphoramide 53-19-0 54-25-1, 6-Azauridine 54-91-1, Pipobroman
55-86-7, Nitrogen mustard 55-98-1, Busulfan 57-13-6, Urea, biological
studies 57-22-7, Vincristine 59-05-2, Methotrexate 60-92-4
115-02-6, Azaserine 147-94-4, Cytosine-arabinoside 148-82-3, Melphalan
154-42-7, 6-Thioguanine 154-93-8, BCNU 157-03-9, DON 303-47-9,
Ochratoxin A 305-03-3, Chlorambucil 320-67-2, 5-Azacytidine
446-86-6, Azathioprine 461-89-2, 6-Azauracil 518-28-5 519-23-3
526-31-8, Abrine??? 671-16-9, Procarbazine 762-03-8 .865-21-4,

Vinblastine 1402-38-6, Actinomycin 1404-00-8, Mitomycin 3778-73-2,
Ifosfamide 4342-03-4, Dacarbazine 5536-17-4, Arabinosyladenine
7440-06-4D, Platinum, antitumor agents-contg. 7665-99-8, Cyclic
guanosine monophosphate 7722-84-1, Hydrogen peroxide, biological studies
7782-44-7, biological studies 9001-01-8, Kininogenase 9001-05-2,
Catalase 9001-12-1, Collagenase 9001-45-0, Glucuronidase 9001-54-1,
Hyaluronidase 9001-62-1, Lipase 9001-99-4, Ribonuclease 9003-98-9,
Desoxyribonuclease 9004-06-2, Elastase 9012-33-3 9013-05-2
9013-66-5 9013-93-8, Phospholipase 9025-82-5, Phosphodiesterase
9027-41-2, Hydrolase 9027-52-5, Hexosaminidase 9031-96-3, Peptidase
9033-33-4, Nucleotidase 9037-29-0, Oxygenase 9054-89-1, Superoxide
dismutase 9055-15-6 9068-67-1, Sulfatase 10028-15-6, Ozone,
biological studies 10048-13-2, Sterigmatocystine 11056-06-7, Bleomycin
11062-77-4, Superoxide anion 13010-47-4, Lomustine 13909-09-6,
Methyl-CCNU 14769-73-4, Levamisole 17902-23-7, Ftorafur 18378-89-7,
Mithramycin 20830-81-3, Daunomycin 21259-20-1, T2 Toxin 23205-42-7,
3-Deazauridine 23214-92-8, Adriamycin 24936-38-7 24937-83-5
36703-88-5, Isoprinosin 39391-18-9, Cyclooxygenase 53643-48-4,
Vindesine 59865-13-3, Cyclosporine 117698-12-1
RL: BIOL (Biological study)
(stimulation of defenses of biol. systems using)

=> d 100:46971 all

ANSWER 1 CAPLUS COPYRIGHT 2003 ACS
AN 1984:46971 CAPLUS
DN 100:46971
TI Synthesis and biological activity studies of selected organophosphorus
esters
AU McElroy, Roger D.; Chambers, Howard W.
CS Dep. Entomol., Mississippi State Univ., Mississippi State, MS, 39762, USA
SO Journal of Agricultural and Food Chemistry (1984), 32(1), 119-23
CODEN: JAFCAU; ISSN: 0021-8561
DT Journal
LA English
CC 5-4 (Agrochemical Bioregulators)
AB Thirty organophosphorus esters (structurally similar DEF analogs) were
synthesized and evaluated as possible insecticide (methyl paraoxon
[950-35-6]) synergists against boll weevils, *Anthonomus grandis*.
B-esterase [9016-18-6] and acetylcholinesterase activity from
organophosphorus-susceptible weevils were measured spectrophotometrically
with S-Ph thiobenzoate and acetylthiocholine as substrates. The
structure-biol. activity relation may be divided into 3 major effects,
i.e., a lipophilic effect, an electronic effect, and a steric effect. In
vitro and in vivo inhibition and toxicity data support the hypothesis that
synergism of Me paraoxon results from the inhibition of the esterase
hydrolyzing S-Ph thiobenzoate by selected organophosphorus esters.
ST insecticide synergist boll weevil methyl paraoxon; phosphorotriithioate
insecticide synergist
IT Insecticides
(esterase-inhibiting, synergists for, tributylphosphorotriithioate
analog as)
IT *Anthonomus grandis*
(insecticide synergists against, tributylphosphorotriithioate analogs as)
IT Molecular structure-biological activity relationship
(insecticidal synergistic, of tributylphosphorotriithioate analogs)
IT 9016-18-6
RL: PROC (Process)
(inhibition of, by insecticide, organophosphorus ester synergists in)
IT 78-48-8P 1642-44-0P 2797-64-0P 3819-72-5P 4081-23-6P 24067-01-4P
24067-02-5P 26115-85-5P 26115-86-6P 30299-04-8P 68598-35-6P

68598-36-7P 68598-37-8P 68598-38-9P 68598-39-0P 68598-40-3P
68598-41-4P 68598-42-5P 78788-15-5P 85480-01-9P 85480-02-0P
85480-03-1P 85480-04-2P 85480-05-3P 85480-06-4P 85480-07-5P
85480-08-6P 85480-09-7P 85480-10-0P 85480-11-1P

RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. and insecticide synergistic activity of, against boll weevil)

IT 950-35-6

RL: BIOL (Biological study)
(tridecylphosphorothioate analogs as insecticidal synergist of,
against bollweevils)

=> d 92:53312 all

ANSWER 1 CAPLUS COPYRIGHT 2003 ACS

AN 1980:53312 CAPLUS

DN 92:53312

TI Biologically active components of anise: toxicity and interactions with
insecticides in insects

AU Marcus, Craig; Lichtenstein, E. Paul

CS Dep. Entomol., Univ. Wisconsin, Madison, WI, 53706, USA

SO Journal of Agricultural and Food Chemistry (1979), 27(6), 1217-23
CODEN: JAFCAU; ISSN: 0021-8561

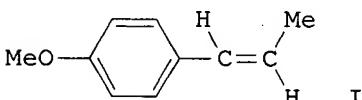
DT Journal

LA English

CC 5-4 (Agrochemicals)

Section cross-reference(s): 12, 62

GI



AB The biol. activity of components of anise tops was studied with insects. trans-Anethole (I) [4180-23-8] was the major insecticidal agent present in anise oil (56% by wt.) derived from anise tops, with an LD₅₀ of 75 .mu.g/fly when topically applied to houseflies. The toxicity of 9 other anise components (anisaldehyde [50984-52-6], estragole [140-67-0], anisyl alc. [1331-81-3], anisic acid [1335-08-6], p-cresol [106-44-5], p-creosol, eugenol [97-53-0], hydroquinone [123-31-9] and acetaldehyde [75-07-0]) to houseflies was also studied. Both anethole [104-46-1] and anisaldehyde increased the toxicity to houseflies when applied simultaneously with parathion [56-38-2], paraoxon [311-45-5], carbaryl [63-25-2], carbofuran [1563-66-2], DDT [50-29-3], or pyrethrum. Also, anethole fed to houseflies as 0.5% of their diet resulted in increased insect mortalities due to topically applied parathion or paraoxon in comparison to flies fed a diet without anethole. Further expts. with houseflies fed anethole as part of their diet indicated that the increased toxicity of paraoxon resulted apparently from an increased penetration of the insecticide into the insect body, and a retardation of its degrdn. to nontoxic, water-sol. metabolites.

ST anise insecticide synergism; anethole insecticide synergism

IT Anise

(anethole from, insecticide synergistic activity and insect metab. of)

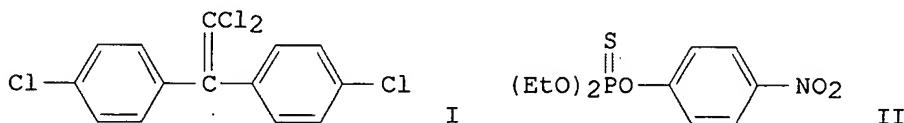
IT Insecticides

Pyrethrins and Pyrethroids

IT RL: BIOL (Biological study)
(anise extractives as synergists of)
Housefly
(anise extractives metab. by, control in relation to)
IT 50-29-3, biological studies 56-38-2 63-25-2 311-45-5 1563-66-2
RL: BIOL (Biological study)
(anise extractives as synergists of)
IT 104-46-1
RL: BIOL (Biological study)
(insect metab. and insecticidal synergistic activity of)
IT 4180-23-8
RL: BOC (Biological occurrence); BSU (Biological study, unclassified);
BIOL (Biological study); OCCU (Occurrence)
(of anise)
IT 75-07-0, biological studies 97-53-0 106-44-5, biological studies
123-31-9, biological studies 140-67-0 1331-81-3 1335-08-6
RL: AGR (Agricultural use); BAC (Biological activity or effector, except
adverse); BSU (Biological study, unclassified); BIOL (Biological study);
USES (Uses)
(of anise, insecticidal activity of)
IT 50984-52-6
RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); BIOL (Biological study)
(of anise, insecticide synergistic activity of)

=> d 86:166109 all

ANSWER 1 CAPLUS COPYRIGHT 2003 ACS
AN 1977:166109 CAPLUS
DN 86:166109
TI DDE increases the toxicity of parathion to *Coturnix quail*
AU Ludke, J. Larry
CS Patuxent Wildl. Res. Cent., U. S. Fish. Wildl. Serv., Laurel, MD, USA
SO Pesticide Biochemistry and Physiology (1977), 7(1), 28-33
CODEN: PCBPBS; ISSN: 0048-3575
DT Journal
LA English
CC 4-4 (Toxicology)
GI



AB Adult male Japanese quail (*C. coturnix*) were exposed to DDE (I) [72-55-9] or chlordane [12789-03-6] in the diet and subsequently dosed with parathion (II) [56-38-2] or paraoxon [311-45-5]. Pretreatment with 5 or 50 ppm I in the diet for 12 weeks resulted in increased cholinesterase (ChE) [9001-08-5] activity in plasma, but not in the brain. Dietary concns. of 5 and 50 ppm I caused increased susceptibility of quail that were challenged with II or paraoxon. The increased mortality resulting from I pretreatment was reflected in brain ChE inhibition. The synergistic action of I was apparent after 3 days of exposure to 50 ppm I and 1 week of exposure to 5 ppm I. Birds exposed for 3 weeks to 5 or 50 ppm I retained their I-potentiated sensitivity to parathion after 2 weeks on clean diet. Chlordane pretreatment resulted in decreased susceptibility (antagonism) to II, but not to paraoxon dosage.

Implications of differing responses in ChE and mortality among controls, I-, and chlordane-pretreated birds after II or paraoxon dosage are discussed.

ST DDE quail parathion toxicity; chlordane quail parathion toxicity; paraoxon toxicity DDE chlordane quail
IT Brain, composition
(cholinesterase of, parathion and paraoxon effect on, in Japanese quail, chlordane and DDE in relation to)
IT Coturnix coturnix
(parathion and paraoxon toxicity to, chlordane and DDE effect on)
IT 9001-08-5
RL: BIOL (Biological study)
(of brain, parathion and paraoxon effect on, in Japanese quail, chlordane and DDE in relation to)
IT 72-55-9, biological studies 12789-03-6
RL: BIOL (Biological study)
(parathion and paraoxon toxicity to Japanese quail response to)
IT 56-38-2 311-45-5
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(toxicity of, to Japanese quail, chlordane and DDE effect on)

=> s stavudine

L1 882 STAVUDINE

=> e esterase

E1 1 ESTERAS/BI
E2 1 ESTERASC/BI
E3 28490 --> ESTERASE/BI
E4 1 ESTERASE1/BI
E5 1 ESTERASE2/BI
E6 1 ESTERASE3/BI
E7 1 ESTERASE5/BI
E8 1 ESTERASE6/BI
E9 1 ESTERASEAMIDASE/BI
E10 1 ESTERASECATALYZED/BI
E11 4 ESTERASELIKE/BI
E12 2 ESTERASELIPASE/BI

=> s e3

L2 28490 ESTERASE/BI

=> s 11 and 12

L3 5 L1 AND L2

=> d 13 1-5

L3 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2003 ACS
AN 2001:454452 CAPLUS
DN 135:313108
TI In vivo pharmacokinetics and metabolism of anti-human immunodeficiency virus agent d4T-5'-(P-bromophenyl methoxyalaninyl phosphate) (sampidine) in mice
AU Chen, Chun-Lin; Venkatachalam, T. K.; Zhu, Zhao-Hai; Uckun, Fatih M.
CS Drug Discovery Program, Department of Pharmaceutical Sciences, Parker Hughes Institute, St. Paul, MN, 55113, USA
SO Drug Metabolism and Disposition (2001), 29(7), 1035-1041
CODEN: DMDSAI; ISSN: 0090-9556
PB American Society for Pharmacology and Experimental Therapeutics
DT Journal
LA English

RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2003 ACS
AN 2000:311663 CAPLUS
DN 133:114591
TI Phosphoramidate derivatives of **stavudine** as inhibitors of HIV-2: unnatural amino acids may substitute for alanine
AU McGuigan, Christopher; Bidois, Laure; Hiouni, Aziz; Ballatore, Carlo; De Clercq, Erik; Balzarini, Jan
CS Welsh School of Pharmacy, Cardiff University, Cardiff, CF1 3XF, UK
SO Antiviral Chemistry & Chemotherapy (2000), 11(2), 111-116
CODEN: ACCHEH; ISSN: 0956-3202
PB International Medical Press
DT Journal
LA English
RE.CNT 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2003 ACS
AN 1998:807805 CAPLUS
DN 130:177179
TI Synthesis, anti-human immunodeficiency virus activity and **esterase** lability of some novel carboxylic ester-modified phosphoramidate derivatives of **stavudine** (d4T)
AU McGuigan, C.; Sutton, P. W.; Cahard, D.; Turner, K.; O'Leary, G.; Wang, Y.; Gumbleton, M.; De Clercq, E.; Balzarini, J.
CS Welsh School Pharmacy, Cardiff University, Cardiff, CF1 3XF, UK
SO Antiviral Chemistry & Chemotherapy (1998), 9(6), 473-479
CODEN: ACCHEH; ISSN: 0956-3202
PB International Medical Press
DT Journal
LA English
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2003 ACS
AN 1998:205372 CAPLUS
DN 128:289775
TI Synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivatives of d4T (**stavudine**): **esterase** hydrolysis as a rapid predictive test for antiviral potency
AU McGuigan, C.; Tsang, H.-W.; Sutton, P. W.; De Clercq, E.; Balzarini, J.
CS Welsh School Pharmacy, University Wales Cardiff, Cardiff, CF1 3XF, UK
SO Antiviral Chemistry & Chemotherapy (1998), 9(2), 109-115
CODEN: ACCHEH; ISSN: 0956-3202
PB International Medical Press
DT Journal
LA English

L3 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2003 ACS
AN 1997:228417 CAPLUS
DN 126:271667
TI A rational strategy for the design of anti-hepatitis B virus nucleotide derivatives
AU Perigaud, Christian; Gosselin, Gilles; Imbach, Jean-Louis
CS Laboratoire de Chimie Bioorganique, UMR CNRS 5625, Montpellier, 34095, Fr.
SO Antiviral Therapy (1996), 1(Suppl. 4, Therapies for Viral Hepatitis), 39-46
CODEN: ANTHFA; ISSN: 1359-6535
PB International Medical Press
DT Journal; General Review
LA English

=> d 13 3-5 all

L3 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2003 ACS
AN 1998:807805 CAPLUS
DN 130:177179
TI Synthesis, anti-human immunodeficiency virus activity and **esterase** lability of some novel carboxylic ester-modified phosphoramidate derivatives of **stavudine** (d4T)
AU McGuigan, C.; Sutton, P. W.; Cahard, D.; Turner, K.; O'Leary, G.; Wang, Y.; Gumbleton, M.; De Clercq, E.; Balzarini, J.
CS Welsh School Pharmacy, Cardiff University, Cardiff, CF1 3XF, UK
SO Antiviral Chemistry & Chemotherapy (1998), 9(6), 473-479
CODEN: ACCHEH; ISSN: 0956-3202
PB International Medical Press
DT Journal
LA English
CC 1-5 (Pharmacology)
Section cross-reference(s): 33
AB We report the design, synthesis and antiviral evaluation of a series of lipophilic, masked phosphoramidate derivs. of the anti-human immunodeficiency virus (HIV) nucleoside analog d4T, designed to act as membrane-sol. pro-drug forms for the free nucleotide. In particular, we report a series of 12 novel compds. with systematic variation in the structure of the carboxylate ester function. In order to rationalize the changes in antiviral action with variation of this moiety we applied the recently developed 31P NMR-based assay for carboxyesterase lability to this series. However, no clear pos. correlation emerged, indicating that, at least within this series, factors other than simple **esterase** lability may be the major determinants of antiviral potency.
ST virus immunodeficiency human phosphoramidate deriv. **stavudine** prepn; HIV virus phosphoramidate deriv **stavudine** prepn
IT Antiviral agents
Human immunodeficiency virus 1
(prepn. and anti-HIV virucidal activity and **esterase** lability of carboxylic ester-modified phosphoramidate derivs. of **stavudine**)
IT 9016-18-6, **Esterase**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(pig liver; prepn. and anti-HIV virucidal activity and **esterase** lability of carboxylic ester-modified phosphoramidate derivs. of **stavudine**)
IT 3056-17-5DP, **Stavudine**, derivs. 173070-83-2P 178469-24-4P
184031-34-3P 184031-40-1P 184031-42-3P 220592-56-3P 220592-57-4P
220592-58-5P 220592-59-6P 220592-60-9P 220592-61-0P 220592-62-1P
220592-74-5P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(prepn. and anti-HIV virucidal activity and **esterase** lability of carboxylic ester-modified phosphoramidate derivs. of **stavudine**)
IT 142629-80-9 183370-70-9 220592-63-2 220592-64-3 220592-65-4
220592-66-5 220592-67-6 220592-68-7 220592-69-8 220592-70-1
220592-71-2 220592-72-3 220592-73-4
RL: RCT (Reactant); RACT (Reactant or reagent)
(prepn. and anti-HIV virucidal activity and **esterase** lability of carboxylic ester-modified phosphoramidate derivs. of **stavudine**)
IT 180076-92-0P

RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. and anti-HIV virucidal activity and **esterase** lability
of carboxylic ester-modified phosphoramidate derivs. of
stavudine)

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

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- (2) Balzarini, J; Proceedings of the National Academy of Sciences USA 1996, V93, P7295 CAPLUS
- (3) McGuigan, C; Antiviral Chemistry and Chemotherapy 1998, V9, P109 CAPLUS
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- (5) McGuigan, C; Journal of Medicinal Chemistry 1996, V39, P1748 CAPLUS
- (6) Meier, C; Synthesis Letters 1998, P233 CAPLUS

L3 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2003 ACS

AN 1998:205372 CAPLUS

DN 128:289775

TI Synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivatives of d4T (**stavudine**): **esterase** hydrolysis as a rapid predictive test for antiviral potency

AU McGuigan, C.; Tsang, H.-W.; Sutton, P. W.; De Clercq, E.; Balzarini, J.

CS Welsh School Pharmacy, University Wales Cardiff, Cardiff, CF1 3XF, UK

SO Antiviral Chemistry & Chemotherapy (1998), 9(2), 109-115

CODEN: ACCHEH; ISSN: 0956-3202

PB International Medical Press

DT Journal

LA English

CC 1-5 (Pharmacology)

Section cross-reference(s): 7, 33

AB Novel chain-extended nucleoside phosphoramidates of the anti-human immunodeficiency virus (HIV) drug d4T (**stavudine**) have been prepd. as possible membrane-permeable prodrugs of the bio-active free 5'-monophosphates. Phosphorochloridate chem. gave the target compds. in moderate to high yields, and all materials were fully characterized by spectroscopic and anal. methods. The compds. are related to the previously reported Ph methoxyalaninyl deriv. of d4T, which was shown to be a potent and selective inhibitor of HIV. In this study the amino acid nitrogen and ester moieties were sepd. by methylene spacers of between two and six carbon atoms. In vitro evaluation of these compds. indicated an almost complete lack of anti-HIV activity, the compds. being several orders of magnitude less potent than the corresponding .alpha.-amino acid derivs. The reasons for the virtual lack of anti-HIV activity appear to involve poor enzyme-mediated hydrolysis.

ST nucleoside phosphoramidate anti human immunodeficiency virus

IT Antiviral agents

Human immunodeficiency virus 1

Human immunodeficiency virus 2

(synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))

IT 9016-18-6, **Esterase**

RL: BPR (Biological process); BSU (Biological study, unclassified); CAT (Catalyst use); BIOL (Biological study); PROC (Process); USES (Uses)

(pig liver; synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))

IT 184031-47-8P 205991-44-2P 205991-51-1P 205991-52-2P 205991-53-3P

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)

(synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))

IT 205991-46-4P 205991-47-5P 205991-48-6P 205991-49-7P 205991-50-0P

RL: BPN (Biosynthetic preparation); BIOL (Biological study); PREP (Preparation)

(synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))

IT 770-12-7, Phenyl phosphorodichloride 1926-80-3, 6-Aminocaproic acid methyl ester hydrochloride 3056-17-5, **Stavudine** 3196-73-4, .beta.-Alanine methyl ester hydrochloride 13031-60-2, 4-Aminobutanoic acid methyl ester hydrochloride 17994-94-4, 7-Aminoheptanoic acid methyl ester hydrochloride 29840-56-0, 5-Aminopentanoic acid methyl ester hydrochloride 173070-83-2 173070-84-3

RL: RCT (Reactant); RACT (Reactant or reagent)

(synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))

IT 205991-54-4P 205991-55-5P 205991-56-6P 205991-57-7P 205991-58-8P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)

(synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))

L3 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2003 ACS
AN 1997:228417 CAPLUS

DN 126:271667

TI A rational strategy for the design of anti-hepatitis B virus nucleotide derivatives

AU Perigaud, Christian; Gosselin, Gilles; Imbach, Jean-Louis
CS Laboratoire de Chimie Bioorganique, UMR CNRS 5625, Montpellier, 34095, Fr.
SO Antiviral Therapy (1996), 1(Suppl. 4, Therapies for Viral Hepatitis), 39-46

CODEN: ANTHFA; ISSN: 1359-6535

PB International Medical Press

DT Journal; General Review

LA English

CC 1-0 (Pharmacology)

AB A review with 42 refs. The potential in antiviral chemotherapy of a pronucleotide approach using mononucleoside phosphotriesters which incorporate S-acyl-2-thioethyl (SATE) groups as esterase-labile transient phosphate protectors is discussed in detail. The use of this approach leads to an increase in the antiretroviral activity of two well-established anti-HIV drugs, namely 2',3'-dideoxyadenosine (ddA) and 2',3'-didehydro-2',3'-dideoxythymidine (**stavudine** or d4T). Moreover, in the case of acyclovir, which is currently used as therapeutic agent for the treatment of herpes virus infections, the corresponding bis(SATE) pronucleotides have emerged as potent and selective inhibitors of the hepatitis B virus replication.

ST review hepatitis virucide nucleotide deriv

IT Antiviral agents

Hepatitis B virus

(strategy for design of anti-hepatitis B virus nucleotide derivs.)

IT Nucleotides, biological studies

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(strategy for design of anti-hepatitis B virus nucleotide derivs.)

=> e physostigmine

E1	6	PHYSOSTIGMIN/BI
E2	1	PHYSOSTIGMINAE/BI
E3	6218 -->	PHYSOSTIGMINE/BI
E4	1	PHYSOSTIGMINEANTAGONISM/BI
E5	1	PHYSOSTIGMINEIN/BI
E6	4	PHYSOSTIGMINELIKE/BI

E7 14 PHYSOSTIGMINES/BI
E8 2 PHYSOSTIGMINESALICYLATE/BI
E9 1 PHYSOSTIGMINETOXICITY/BI
E10 1 PHYSOSTIGMINETREATED/BI
E11 1 PHYSOSTIGMINIC/BI
E12 2 PHYSOSTIGMINICO/BI

=> s e3-e7

6218 PHYSOSTIGMINE/BI
1 PHYSOSTIGMINEANTAGONISM/BI
1 PHYSOSTIGMINEIN/BI
4 PHYSOSTIGMINELIKE/BI
14 PHYSOSTIGMINES/BI

L4 6222 (PHYSOSTIGMINE/BI OR PHYSOSTIGMINEANTAGONISM/BI OR PHYSOSTIGMINE
IN/BI OR PHYSOSTIGMINELIKE/BI OR PHYSOSTIGMINES/BI)

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(FILE 'HOME' ENTERED AT 08:30:09 ON 02 JUN 2003)

FILE 'CAPLUS' ENTERED AT 08:30:33 ON 02 JUN 2003

L1 882 S STAVUDINE
E ESTERASE
L2 28490 S E3
L3 5 S L1 AND L2
E PHYSOSTIGMINE
L4 6222 S E3-E7

=> s l2 and l4

L5 274 L2 AND L4

=> s l5 and viral

119945 VIRAL
L6 0 L5 AND VIRAL

=> s l1 and l5

L7 1 L1 AND L5

=> d 17 all

L7 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2003 ACS
AN 2001:454452 CAPLUS
DN 135:313108

TI In vivo pharmacokinetics and metabolism of anti-human immunodeficiency
virus agent d4T-5'-[P-bromophenyl methoxyalaninyl phosphate] (sampidine)
in mice

AU Chen, Chun-Lin; Venkatachalam, T. K.; Zhu, Zhao-Hai; Uckun, Fatih M.
CS Drug Discovery Program, Department of Pharmaceutical Sciences, Parker

Hughes Institute, St. Paul, MN, 55113, USA

SO Drug Metabolism and Disposition (2001), 29(7), 1035-1041
CODEN: DMDSAI; ISSN: 0090-9556

PB American Society for Pharmacology and Experimental Therapeutics

DT Journal

LA English

CC 1-2 (Pharmacology)

AB D4T-5'-[p-Sampidine, bromophenyl methoxyalaninyl phosphate] (HI-113), a
novel aryl phosphate deriv. of stavudine (d4T), exhibits
substantially more potent anti-human immunodeficiency virus activity than
d4T. The purpose of the present study was to investigate the in vivo
pharmacokinetics and metab. of this promising new anti-HIV agent in mice.
Here, the authors report that HI-113 forms 2 active metabolites with
favorable pharmacokinetics after systemic administration. Plasma HI-113

concns. were measured by anal. high-performance liq. chromatog. and the pharmacokinetic parameters were estd. using the WinNonlin program. After i.v. administration, the elimination half-life ($t_{1/2}$) of HI-113 was 3.6 min with a systemic clearance of 174.5 mL/min/kg. HI-113 was converted to the active metabolites alaninyl-d4T-monophosphate (ala-d4T-MP) and d4T. The Tmax values for ala-d4T-MP and d4T derived from i.v. administered HI-113 were 5.1 and 17.4 min, resp. The elimination half-life for synthetic ala-d4T-MP was 38.9 min after i.v. administration. Ala-d4T-MP was metabolized to form d4T (Tmax = 5.0 min). The elimination half-life of d4T derived from i.v. administered ala-d4T-MP (32.4 min) was similar to the elimination half-life of i.v. administered d4T (26.6 min). In contrast, the elimination half-life of d4T derived from HI-113 was substantially longer (116.2 min). Similarly, the elimination half-life of ala-d4T-MP derived from HI-113 (138.8 min) was markedly longer than the elimination half-life of ala-d4T-MP given i.v. (38.9 min). Following oral administration of HI-113, the elimination half-lives of ala-d4T-MP (56.1 min) and d4T (102.6 min) were also prolonged.

ST antiHIV agent sampilidine pharmacokinetics; HI113 antiHIV agent pharmacokinetics
IT Anti-AIDS agents
(in vivo pharmacokinetics and metab. of anti-human immunodeficiency virus agent d4T-5'-(P-bromophenyl methoxyalaninyl phosphate) in mice)
IT 57-47-6, **Physostigmine** 60-00-4, EDTA, biological studies
311-45-5
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(effect of esterase inhibitors on HI-113 metab. in plasma)
IT 3056-17-5, d4T 180076-92-0
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)
(in vivo pharmacokinetics and metab. of anti-human immunodeficiency virus agent d4T-5'-(P-bromophenyl methoxyalaninyl phosphate) in mice)
IT 217178-62-6, Sampidine
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(in vivo pharmacokinetics and metab. of anti-human immunodeficiency virus agent d4T-5'-(P-bromophenyl methoxyalaninyl phosphate) in mice)
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Augustinsson, K; Ann NY Acad Sci 1961, V94, P884
(2) Balzarini, J; Mol Pharmacol 1996, V50, P1207 CAPLUS
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(FILE 'HOME' ENTERED AT 08:30:09 ON 02 JUN 2003)

FILE 'CAPLUS' ENTERED AT 08:30:33 ON 02 JUN 2003

L1 882 S STAVUDINE
E ESTERASE
L2 28490 S E3
L3 5 S L1 AND L2
E PHYSOSTIGMINE
L4 6222 S E3-E7
L5 274 S L2 AND L4
L6 0 S L5 AND VIRAL
L7 1 S L1 AND L5

=> d 15 260-274

L5 ANSWER 260 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1939:64889 CAPLUS
DN 33:64889
OREF 33:9333b-d
TI Enzymic hydrolysis of acetylneurine
AU Massart, L.; Dufait, R.
SO Naturwissenschaften (1939), 27, 567
DT Journal
LA Unavailable

L5 ANSWER 261 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1939:14855 CAPLUS
DN 33:14855
OREF 33:2218e-f
TI The effect of drugs on cholinesterase
AU Keeser, Ed.
SO Klin. Wochschr. (1938), 17, 1811
DT Journal
LA Unavailable

L5 ANSWER 262 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1939:1202 CAPLUS
DN 33:1202
OREF 33:184i,185a-b
TI The hydrolysis of homatropine and atropine by various tissues
AU Bernheim, Frederick; Bernheim, Mary L. C.
SO J. Pharmacol. (1938), 64, 209-16
DT Journal
LA Unavailable

L5 ANSWER 263 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1938:9241 CAPLUS
DN 32:9241
OREF 32:1337g-h

TI Determination of the antagonism between curare, metrazole and coramine
AU Emmelin, N.; Kahlson, G.
SO Skand. Arch. Physiol. (1937), 77, 312-18
DT Journal
LA Unavailable

L5 ANSWER 264 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1937:53993 CAPLUS
DN 31:53993
OREF 31:7509e-g
TI Some recent extensions of chemical transmission
AU Dale, H. H.
SO Cold Springs Harbor Symposia Quant. Biol. (1936), 4, 143-9
DT Journal
LA Unavailable

L5 ANSWER 265 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1937:19070 CAPLUS
DN 31:19070
OREF 31:2674i, 2675a
TI From **physostigmine** to prostigmine
AU Barger, George
SO Festschrift Emil C. Barell (1936) 7-17
DT Journal
LA Unavailable

L5 ANSWER 266 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1936:62657 CAPLUS
DN 30:62657
OREF 30:8350d-g
TI Recent advances in knowledge concerning the chemical mediation of nerve
impulses
AU Butt, H. R.
SO Proc. Staff Meetings Mayo Clinic (1936), 11, 327-31
DT Journal
LA Unavailable

L5 ANSWER 267 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1936:48386 CAPLUS
DN 30:48386
OREF 30:6449i
TI Synergism of **physostigmine** and acetylcholine
AU Freud, J.; Uyldert, Ina E.
SO Arch. intern. pharmacodynamic (1936), 52, 238-44
DT Journal
LA Unavailable

L5 ANSWER 268 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1936:8466 CAPLUS
DN 30:8466
OREF 30:1124a-d
TI A theory of the sensitization to acetylcholine, and the effect of fluoride
in raising the irritability
AU Kahlson, G.; Uvnas, B.
SO Skand. Arch. Physiol. (1935), 72, 215-39
DT Journal
LA Unavailable

L5 ANSWER 269 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1936:856 CAPLUS
DN 30:856
OREF 30:124b-d

TI The esterase activity of human blood plasma
AU Vahlquist, Bo
SO Skand. Arch. Physiol. (1935), 72, 133-60
DT Journal
LA Unavailable

L5 ANSWER 270 OF 274 CAPIUS COPYRIGHT 2003 ACS
AN 1935:36782 CAPIUS
DN 29:36782
OREF 29:4780h-i
TI The acetylcholine-destroying action of blood
AU Ammon, R.; Voss, G.
SO Arch. ges. Physiol. (Pflugers) (1935), 235, 393-400
DT Journal
LA Unavailable

L5 ANSWER 271 OF 274 CAPIUS COPYRIGHT 2003 ACS
AN 1933:7569 CAPIUS
DN 27:7569
OREF 27:776i,777a
TI Pharmacological studies on the leech preparation as well as a method for the biological demonstration of acetylcholine in the presence of other pharmacologically active substances of body origin
AU Minz, B.
SO Arch. exptl. Path. Pharmakol. (1932), 168, 292-304
DT Journal
LA Unavailable

L5 ANSWER 272 OF 274 CAPIUS COPYRIGHT 2003 ACS
AN 1930:44686 CAPIUS
DN 24:44686
OREF 24:4840c-d
TI Fermentative splitting of acetylcholine in blood and its inhibition by **physostigmine**
AU Engelhart, E.; Loewi, O.
SO Arch. exptl. Path. u. Pharm. (1930), 150, 1-13
DT Journal
LA Unavailable

L5 ANSWER 273 OF 274 CAPIUS COPYRIGHT 2003 ACS
AN 1927:10652 CAPIUS
DN 21:10652
OREF 21:1300f-h
TI The vagus substance
AU Loewi, O.
SO Naturwissenschaften (1920), 14, 994-5
DT Journal
LA Unavailable

L5 ANSWER 274 OF 274 CAPIUS COPYRIGHT 2003 ACS
AN 1927:9372 CAPIUS
DN 21:9372
OREF 21:1144a-b
TI Humoral transfer of heat-nerve action. XI. Mechanism of the action of **physostigmine** and of ergotamine on vagus action
AU Loewi, O.; Navratil, E.
SO Arch. ges. Physiol. (Pfluger's) (1926), 214, 689-96
DT Journal
LA Unavailable

=> d 15 269 261 all

L5 ANSWER 269 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1936:856 CAPLUS
DN 30:856
OREF 30:124b-d
TI The **esterase** activity of human blood plasma
AU Vahlquist, Bo
SO Skand. Arch. Physiol. (1935), 72, 133-60
DT Journal
LA Unavailable
CC 11A (Biological Chemistry: General)
AB To decide whether human plasma contains a specific choline **esterase** or the hydrolysis is brought about by the ordinary lipase, a study was made by various methods. Cataphoretically both activities moved strictly parallel in the elec. field and independently of the migration of the albumin and globulin. Similarly quinine, atoxyl and **physostigmine** inhibited the action of the **esterase** no matter what substrate was employed (acetylcholine, tributyrin or Me butyrate). Parallel detns. of choline and tributyrin **esterase** activity were made on different individuals under a great variety of conditions. The correlation of all these results was so great that the correlation coeff. was 0.92 +- 0.02. All 3 modes of attack on this problem indicate, therefore, that there is no specific acetylcholine **esterase**. The **esterase** content is not appreciably affected by ingestion of food, muscular exercise, nervous excitement, menstruation or pregnancy. Under conditions of abnormal muscular spasms such as bronchial asthma or ulcus ventriculi the values are relatively low but still within the normal range. Only in tuberculosis is the **esterase** content abnormally low. The **esterase** apparently can only act to protect the organism against an accumulation of acetylcholine in the blood.

L5 ANSWER 261 OF 274 CAPLUS COPYRIGHT 2003 ACS
AN 1939:14855 CAPLUS
DN 33:14855
OREF 33:2218e-f
TI The effect of drugs on cholinesterase
AU Keeser, Ed.
SO Klin. Wochschr. (1938), 17, 1811
DT Journal
LA Unavailable
CC 11H (Biological Chemistry: Pharmacology)
AB Glutathione, sympathol and pilocarpine activated cholinesterase in vitro; atropine, **physostigmine**, prostigmine, cocaine, hordenine and muscarine inhibited the **esterase**.

=> d his

(FILE 'HOME' ENTERED AT 08:30:09 ON 02 JUN 2003)

FILE 'CAPLUS' ENTERED AT 08:30:33 ON 02 JUN 2003

L1 882 S STAVUDINE
E ESTERASE
L2 28490 S E3
L3 5 S L1 AND L2
E PHYSOSTIGMINE
L4 6222 S E3-E7
L5 274 S L2 AND L4
L6 0 S L5 AND VIRAL
L7 1 S L1 AND L5

=> e paraoxon

E1 1 PARAOXIN/BI
E2 1 PARAOXINASE/BI
E3 2427 --> PARAOXON/BI
E4 652 PARAOXONASE/BI
E5 4 PARAOXONASE1/BI
E6 1 PARAOXONASE2/BI
E7 1 PARAOXONASEE/BI
E8 25 PARAOXONASES/BI
E9 18 PARAOXONE/BI
E10 1 PARAOXONETHYL/BI
E11 1 PARAOXONHYDROLYZING/BI
E12 1 PARAOXONIC/BI

=> s e3

L8 2427 PARAOXON/BI

=> s 18 and 12

L9 434 L8 AND L2

=> d 19 400-434

L9 ANSWER 400 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1970:29245 CAPLUS
DN 72:29245
TI **Esterase** activity in organophosphorus-tolerant strains of *Aedes aegypti*
AU Ziv, M.; Brown, Anthony W. A.
CS Univ. Western Ontario, London, ON, Can.
SO Mosquito News (1969), 29(3), 456-61
CODEN: MOSQAU; ISSN: 0027-142X
DT Journal
LA English

L9 ANSWER 401 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1970:1782 CAPLUS
DN 72:1782
TI Delayed neurotoxic effect of some organophosphorus compounds.
Identification of the phosphorylation site as an **esterase**
AU Johnson, Martin Keith
CS Med. Res. Counc. Lab., Carshalton, UK
SO Biochemical Journal (1969), 114(4), 711-17
CODEN: BIJOAK; ISSN: 0264-6021
DT Journal
LA English

L9 ANSWER 402 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1969:477576 CAPLUS
DN 71:77576
TI Organophosphate inhibitors of acetylcholine-receptor and **-esterase**
tested on the electroplax
AU Bartels, Eva; Nachmansohn, David
CS Coll. of Phys. and Surg., Columbia Univ., New York, NY, USA
SO Archives of Biochemistry and Biophysics (1969), 133(1), 1-10
CODEN: ABBIA4; ISSN: 0003-9861
DT Journal
LA English

L9 ANSWER 403 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1969:420623 CAPLUS
DN 71:20623
TI Effects of drugs on the uptake of acetylcholine in rat brain cortex slices

AU Liang, C. C.; Quastel, J. H.
CS Univ. British Columbia, Vancouver, Can.
SO Biochemical Pharmacology (1969), 18(5), 1187-94
CODEN: BCPCA6; ISSN: 0006-2952
DT Journal
LA English

L9 ANSWER 404 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1969:85286 CAPLUS
DN 70:85286
TI Quinone and hydrocarbon production in the defensive glands of *Eleodes longicollis* and *Tribolium castaneum*
AU Happ, George M.
CS Cornell Univ., Ithaca, NY, USA
SO Journal of Insect Physiology (1968), 14(12), 1821-37
CODEN: JIPHAF; ISSN: 0022-1910
DT Journal
LA English

L9 ANSWER 405 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1969:56022 CAPLUS
DN 70:56022
TI Gastrointestinal absorption of the **esterase**-reactivating substance obidoxime and the possibility of facilitating its absorption
AU Erdmann, Wolf D.; Okonek, S.
CS Inst. Pharmakol. Toxikol., Univ. Goettingen, Goettingen, Fed. Rep. Ger.
SO Archiv fuer Toxikologie (1969), 24(2), 91-101
CODEN: ATXKA8; ISSN: 0003-9446
DT Journal
LA German

L9 ANSWER 406 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1969:45050 CAPLUS
DN 70:45050
TI Direct measurement of acetylesterase in living protist cells
AU Medzon, Edward L.; Brady, Marilyn L.
CS Univ. Western Ontario, London, ON, Can.
SO Journal of Bacteriology (1969), 97(1), 402-15
CODEN: JOBAAY; ISSN: 0021-9193
DT Journal
LA English

L9 ANSWER 407 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1969:17208 CAPLUS
DN 70:17208
TI An enzyme in hen brain hydrolyzing phenyl phenylacetate: possible connection with the delayed neurotoxic effect of some organophosphorus compounds
AU Johnson, Martin Keith
CS Med. Res. Counc. Toxicol. Res. Unit, Carshalton, UK
SO Biochemical Journal (1968), 110(2), 13P
CODEN: BIJOAK; ISSN: 0264-6021
DT Journal
LA English

L9 ANSWER 408 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1969:10279 CAPLUS
DN 70:10279
TI Inhibition of brain acetylcholinesterase by organophosphorus and carbamate compounds
AU Kosugi, Yoshihiro
CS Osaka City Univ. Grad. Sch., Osaka, Japan

SO Osaka-shiritsu Daigaku Igaku Zasshi (1968), 17(1-2), 3-15
CODEN: OSDIAF; ISSN: 0472-1446
DT Journal
LA Japanese

L9 ANSWER 409 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1968:459055 CAPLUS
DN 69:59055
TI Oximes of 1-benzoyl- and 1-phenacylpyridinium chloride and 1-phenyl-, 1-benzyl-, 1-benzoyl-, and 1-phenacyl-4-formylpyridinium chloride. Synthesis and biochemical significance
AU Binenfeld, Zlatko; Milojevic, Miloje M.; Milosevic, Milenko P.; Andelkovic, Draginja I.
CS Inst. Pharmacol., Belgrade
SO Glasnik Hemijskog Drustva Beograd (1966), 31(4-6), 243-50
CODEN: GHDBAX; ISSN: 0017-0941
DT Journal
LA Serbian

L9 ANSWER 410 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1967:442617 CAPLUS
DN 67:42617
TI Pesticide residues in food
AU Koivistoinen, Pekka
SO Kemian Teollisuus (1967), 24(1), 23-7
CODEN: KETEA9; ISSN: 0022-9822
DT Journal
LA Finnish

L9 ANSWER 411 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1966:430869 CAPLUS
DN 65:30869
OREF 65:5753a-c
TI Purification and characterization of a proteinase excreted by Calliphora erythrocephala larvae
AU Moser, Joerg G.
CS Freie Univ., Berlin
SO Biochemische Zeitschrift (1966), 344(4), 337-52
CODEN: BIZEA2; ISSN: 0366-0753
DT Journal
LA German

L9 ANSWER 412 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1965:501084 CAPLUS
DN 63:101084
OREF 63:18659e-f
TI Readily volatile terpenes and terpene mixtures (essential oils) as carriers of alleopathic effects. II. Hydrolysis of methyl acetate by cress seedlings
AU Hefendehl, F. W.
CS Univ. Freiburg/Br., Germany
SO Flora (Jena) (1965), 156(2), 173-6
DT Journal
LA German

L9 ANSWER 413 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1965:38853 CAPLUS
DN 62:38853
OREF 62:6881e-h
TI The histochemistry of the esterase of mast cells
AU Keller, R.
CS Dermatol. Universitatsklin., Zurich, Switz.

SO Schweizerische Medizinische Wochenschrift (1963), 93, 1504-5
CODEN: SMWOAS; ISSN: 0036-7672

DT Journal
LA German

L9 ANSWER 414 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1961:100873 CAPLUS

DN 55:100873

OREF 55:19005c-e

TI In vitro inhibition and reactivation of cholinesterases following para-oxon and DFP poisoning

AU Latki, O.; Erdmann, W. D.

CS Univ. Gottingen, Germany

SO Naunyn-Schmiedebergs Archiv fuer Experimentelle Pathologie und Pharmakologie (1961), 240, 514-22

CODEN: AEPPAE; ISSN: 0365-2009

DT Journal

LA Unavailable

L9 ANSWER 415 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1961:89087 CAPLUS

DN 55:89087

OREF 55:16835a-c

TI Cholinesterase and aliesterase activity in organophosphorus-susceptible and -resistant houseflies

AU Bigley, Walter S.; Plapp, Frederick W., Jr.

CS U.S. Dept. of Agr., Corvallis, OR

SO Annals of the Entomological Society of America (1960), 53, 360-4

CODEN: AESAAI; ISSN: 0013-8746

DT Journal

LA Unavailable

L9 ANSWER 416 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1960:98916 CAPLUS

DN 54:98916

OREF 54:18799i,18800a-e

TI The interaction between organophosphorus insecticides and esterases in homogenates of organophosphate susceptible and resistant houseflies

AU van Asperen, K.; Oppenoorth, F. J.

CS Lab. Insekticidenonderzoek, Utrecht, Neth.

SO Entomologia Experimentalis et Applicata (1960), 3(No. 1), 68-83

CODEN: ETEAAT; ISSN: 0013-8703

DT Journal

LA English

L9 ANSWER 417 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1960:74852 CAPLUS

DN 54:74852

OREF 54:14316d-h

TI Differentiation of the A-type esterases in sheep serum

AU Main, A. R.

CS Univ. Cambridge, UK

SO Biochemical Journal (1960), 75, 188-95

CODEN: BIJOAK; ISSN: 0264-6021

DT Journal

LA Unavailable

L9 ANSWER 418 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1959:64079 CAPLUS

DN 53:64079

OREF 53:11656h-i,11657a

TI Unspecific paralyzing action of some alkyl phosphates

AU Erdmann, W. D.; Sakai, F.
CS Univ. Gottingen, Germany
SO Naunyn-Schmiedebergs Archiv fuer Experimentelle Pathologie und Pharmakologie (1959), 236, 205-7
CODEN: AEPPAE; ISSN: 0365-2009
DT Journal
LA Unavailable

L9 ANSWER 419 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1959:60411 CAPIUS
DN 53:60411
OREF 53:10920a-d
TI Chemical studies on insecticides. VI. The rate of hydrolysis of some phosphorus acid esters
AU Ketelaar, J. A. A.; Gersmann, H. R.
CS Univ. Amsterdam
SO Rev. trav. chim. (1958), 77, 973-81
DT Journal
LA English

L9 ANSWER 420 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1958:79156 CAPIUS
DN 52:79156
OREF 52:14068a-c
TI Mode of action of organophosphorus insecticides
AU van Asperen, K.
CS Lab. for Research on Insecticides. T. N. O., Utrecht, Neth.
SO Nature (London, United Kingdom) (1958), 181, 355-6
CODEN: NATUAS; ISSN: 0028-0836
DT Journal
LA Unavailable

L9 ANSWER 421 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1958:57648 CAPIUS
DN 52:57648
OREF 52:10413d-f
TI Antagonism between atropine and cholinesterase poisons investigated by a microbiological technique
AU Neubert, Diether; Maibauer, Dieter
CS Freie Univ., Berlin
SO Naunyn-Schmiedebergs Archiv fuer Experimentelle Pathologie und Pharmakologie (1958), 233, 163-72
CODEN: AEPPAE; ISSN: 0365-2009
DT Journal
LA Unavailable

L9 ANSWER 422 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1958:46522 CAPIUS
DN 52:46522
OREF 52:8368i,8369a-b
TI Analysis of the stimulating and paralyzing effects of alkyl phosphates (parathion, paraoxon, systox) tested on the isolated rabbit intestine
AU Erdmann, W. D.; Heye, D.
CS Univ. Gottingen, Germany
SO Naunyn-Schmiedebergs Archiv fuer Experimentelle Pathologie und Pharmakologie (1958), 232, 507-21
CODEN: AEPPAE; ISSN: 0365-2009
DT Journal
LA Unavailable

L9 ANSWER 423 OF 434 CAPIUS COPYRIGHT 2003 ACS

AN 1958:26662 CAPLUS
DN 52:26662
OREF 52:4842f-g
TI Specific antidote treatment in prolonged poisoning with alkylphosphates in guinea pigs
AU Erdmann, W. D.; Schmidt, G.
SO Naunyn-Schmiedebergs Archiv fuer Experimentelle Pathologie und Pharmakologie (1957), 232, 230-2
CODEN: AEPAAE; ISSN: 0365-2009
DT Journal
LA Unavailable

L9 ANSWER 424 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1957:63727 CAPLUS
DN 51:63727
OREF 51:11599g,11600a-b
TI Aromatic esterase in insects
AU Metcalf, R. L.; Maxon, M.; Fukuto, T. R.; March, R. B.
CS Citrus Expt. Sta., Riverside, CA
SO Ann. Entomol. Soc. Am. (1956), 49, 274-9
From: Bee World, 1957, 159(1957)
DT Journal
LA Unavailable

L9 ANSWER 425 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1957:53475 CAPLUS
DN 51:53475
OREF 51:9925d-f
TI Toxicity and elimination of esterase-blocking alkyl phosphates and eserine in prolonged infusions
AU Erdmann, W. D.; Lendle, L.
CS Univ. Gottingen, Germany
SO Arch. exptl. Pathol. Pharmakol., Naunyn-Schmiedeberg's (1957), 230, 208-22
DT Journal
LA Unavailable

L9 ANSWER 426 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1957:48474 CAPLUS
DN 51:48474
OREF 51:9000h-i,9001a-d
TI A specific antidote against lethal alkyl phosphate intoxication. IV. Effects in brain
AU Kewitz, Helmut; Nachmansohn, David
CS Columbia Univ.
SO Arch. Biochem. Biophys. (1957), 66, 271-83
DT Journal
LA Unavailable

L9 ANSWER 427 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1957:48473 CAPLUS
DN 51:48473
OREF 51:9000d-h
TI A specific antidote against lethal alkyl phosphate intoxication. III. Repair of chemical lesion
AU Kewitz, Helmut
CS Columbia Univ.
SO Arch. Biochem. Biophys. (1957), 66, 263-70
DT Journal
LA Unavailable

L9 ANSWER 428 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1957:2720 CAPLUS

DN 51:2720
OREF 51:602f-h
TI Parathion metabolism in rat liver and kidney slices
AU Kubistova, J.
CS Inst. Ind. Hyg. Occupational Diseases, Prague
SO Experientia (1956), 12, 333-5
DT Journal
LA English

L9 ANSWER 429 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1956:41827 CAPIUS
DN 50:41827
OREF 50:8072d-f
TI The role of A-esterase in the acute toxicity of **paraoxon**, TEPP, and Parathion
AU Main, A. R.
CS Dept. Natl. Health and Welfare, Ottawa
SO Canadian Journal of Biochemistry and Physiology (1956), 34, 197-216
CODEN: CJBPAZ; ISSN: 0576-5544
DT Journal
LA Unavailable

L9 ANSWER 430 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1956:33753 CAPIUS
DN 50:33753
OREF 50:6736i,6737a-b
TI Insecticidal and antiesterase activity of organophosphorus compounds
AU Lord, K. A.; Potter, Chas.
CS Rothamsted Exptl. Sta., Harpenden, Herts, UK
SO Chemistry & Industry (London, United Kingdom) (1954) 1214-17
CODEN: CHINAG; ISSN: 0009-3068
DT Journal
LA Unavailable

L9 ANSWER 431 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1955:9787 CAPIUS
DN 49:9787
OREF 49:2010a-c
TI Differences in esterases from insect species: toxicity of organophosphorus compounds and in vitro antiesterase activity
AU Lord, K. A.; Potter, C.
CS Rothamsted Exptl. Sta., Harpenden, UK
SO Journal of the Science of Food and Agriculture (1954), 5, 490-8
CODEN: JSFAAE; ISSN: 0022-5142
DT Journal
LA Unavailable

L9 ANSWER 432 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1954:68408 CAPIUS
DN 48:68408
OREF 48:12205c-e
TI Enzymic effects of rabbit serum
AU Mounter, L. A.
CS Univ. of Virginia, Charlottesville
SO J. Biol. Chem. (1954), 209, 813-17
DT Journal
LA Unavailable

L9 ANSWER 433 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1954:61546 CAPIUS
DN 48:61546
OREF 48:10934a-c

TI Acetylcholine production in animals poisoned by diethyl p-nitrophenyl phosphate (**paraoxon**)
AU Barnes, J. M.; Duff, Janet I.
CS Med. Research Council, Carshalton Beeches, Surrey, UK
SO Brit. J. Pharmacol. (1954), 9, 153-8
DT Journal
LA Unavailable

L9 ANSWER 434 OF 434 CAPLUS COPYRIGHT 2003 ACS

AN 1954:56910 CAPLUS

DN 48:56910

OREF 48:10082d-g

TI Inhibition of trypsin and chymotrypsin by certain organic phosphorus esters
AU Kilby, B. A.; Youatt, G.
CS 9 Hyde Terrace, Leeds, UK
SO Biochem. J. (1954), 57, 303-9
DT Journal
LA Unavailable

=> d 19 430 432 422 413 all

L9 ANSWER 430 OF 434 CAPLUS COPYRIGHT 2003 ACS

AN 1956:33753 CAPLUS

DN 50:33753

OREF 50:6736i,6737a-b

TI Insecticidal and antiesterase activity of organophosphorus compounds
AU Lord, K. A.; Potter, Chas.
CS Rothamsted Exptl. Sta., Harpenden, Herts, UK
SO Chemistry & Industry (London, United Kingdom) (1954) 1214-17
CODEN: CHINAG; ISSN: 0009-3068

DT Journal

LA Unavailable

CC 15A (Pesticides and Crop-Control Agents)

AB cf. C.A. 47, 259c. The possibility that the primary toxic action of organophosphorus compds. may not be on esterases hydrolyzing acetylcholine (I) but on other esterases was suggested by detn. of the amt. of I or PhOAc hydrolyzed by exts. of 7 species of whole insects. In several species (e.g. *Dysdercus fasciatus* adult males, *Tenebrio molitor* adults and larvae) the hydrolysis of PhOAc was markedly greater than I. Inhibition of hydrolysis by tetraethyl pyrophosphate, **paraoxon**, parathion, O,S-diethyl O-(p-nitrophenyl) thiophosphate and O,O-diethyl S-(p-nitrophenyl) thiophosphate on 4 species of insects showed that general **esterase** activity is usually more readily inhibited in vitro than PhOAc hydrolysis is, and that the enzymes responsible for both I and PhOAc hydrolysis are not identical from species to species since the concn. of any given inhibitor required to inhibit 50% of the activity differs. The data do not show any correlation between in vitro enzyme inhibition and in vivo toxicity.

IT *Dysdercus fasciatus*

(P-compd. effect on esterases of)

IT Insects

(cholinesterase and esterases of, P compd. effect on)

IT Insecticides

(phosphorus compds. or P-contg., effect on esterases)

IT Benzenethiol, p-nitro-, S-ester with O,O-diethyl phosphorothioate
Ethanethiol, S-ester with O-ethyl O-p-nitrophenyl phosphorothioate
Phenol, p-nitro-, S-ester with O,O-di-Et phosphorothioate
(effect on cholinesterase and non-specific esterases)

IT Phosphoric acid, diethyl p-nitrophenyl ester

Phosphorothioic acid, O,S-diethyl O-p-nitrophenyl ester

IT (effect on cholinesterase and nonspecific esterases)
IT Acetic acid, phenyl ester
 (esterases hydrolyzing, P insecticide effect on)
IT Esterases
 (insect, P-compd. effect on)
IT 7723-14-0, Phosphorus
 (compds., **esterase** response to)
IT 56-38-2, Parathion
 (effect on cholinesterase and non-specific esterases)
IT 107-49-3, Ethyl pyrophosphate, Et4P2O7
 (effect on cholinesterases and nonspecific esterases)
IT 9000-81-1, Acetylcholinesterase
 (esterases hydrolyzing, P insecticide effect on)
IT 485-43-8, Iridomyrmecin
 (prepn. of)

L9 ANSWER 432 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1954:68408 CAPIUS
DN 48:68408
OREF 48:12205c-e
TI Enzymic effects of rabbit serum
AU Mounter, L. A.
CS Univ. of Virginia, Charlottesville
SO J. Biol. Chem. (1954), 209, 813-17
DT Journal
LA Unavailable
CC 11A (Biological Chemistry: General)
AB cf. C.A. 47, 11279c, 12462i; 48, 211g. Previous investigations with rabbit serum showed that diiso-Pr fluophosphate (I), di-Et p-nitrophenyl phosphate (**Paraoxon**) (II), and p-substituted aromatic esters are hydrolyzed. The present evidence indicates that 1 enzyme (an A **esterase**) in rabbit serum hydrolyzes I, tetra-Et pyrophosphate (III), II, and p-O2NC6H4O2CMe. Enzymes which hydrolyze I and III cannot always be classified as A esterases (Aldridge, C.A. 47, 8123i). The rabbit-serum enzyme is inhibited by Mn and Co(II); these ions activate the hog-kidney enzyme (loc. cit.).
IT Enzymes
 (blood-serum, of rabbit)
IT Blood serum
 (enzymes in, of rabbit)
IT Esterases
 (A, in rabbit serum)
IT Isopropyl phosphorofluoridate, (iso-PrO)2FPO
 (hydrolysis of, by A **esterase** of blood serum)
IT 7440-48-4, Cobalt
 (A **esterase** (rabbit-serum) inhibition by)
IT 7439-96-5, Manganese
 (**esterase** A (rabbit-serum) inhibition by)
IT 830-03-5, Phenol, p-nitro-, acetate
 (hydrolysis by A **esterase** of rabbit serum)
IT 107-49-3, Ethyl pyrophosphate, Et4P2O7
 (hydrolysis of, by A **esterase** of rabbit serum)

L9 ANSWER 422 OF 434 CAPIUS COPYRIGHT 2003 ACS
AN 1958:46522 CAPIUS
DN 52:46522
OREF 52:8368i, 8369a-b
TI Analysis of the stimulating and paralyzing effects of alkyl phosphates (parathion, **paraoxon**, systox) tested on the isolated rabbit intestine
AU Erdmann, W. D.; Heye, D.
CS Univ. Gottingen, Germany

SO Naunyn-Schmiedebergs Archiv fuer Experimentelle Pathologie und Pharmakologie (1958), 232, 507-21
CODEN: AEPPAE; ISSN: 0365-2009
DT Journal
LA Unavailable
CC 11H (Biological Chemistry: Pharmacology)
AB The special effects and the min. effective concns. of the 3 drugs are nearly equal. The effects are: an increase in pendular motility and tonus at 2-4 times. 10-8. **Paraoxon** is different from the other substances by causing peristalsis at 2 times. 10-8 to 2 times. 10-6. After washing out the parathion with Ringer soln. the excitation disappears. Washing produces an increase in excitation of the intestine after **paraoxon** and systox. Inhibition of intestinal motility sets in at concns. 1-5 times. 10-5. The paralytic effects are abolished by washing. These effects are the result of inhibition of the intestinal cholinesterase. Points of attack are the parasympathetic receptors. The peristaltic type of motility is the result of inhibition of a crit. quantity of intestinal **esterase** with relation to the autonomic ganglia. The paralytic effect seems to be the result of direct action on the smooth muscle (expts. with physostigmine, BaCl₂, and specific reactivators of **esterase**).
IT Intestines
(effect of parathion, paraoxen and septox on)
IT Phosphoric acid, diethyl p-nitrophenyl ester
(in intestinal stimulation and paralysis)
IT Cholinesterases
(in intestines, inhibition by alkyl phosphates)
IT Phosphorothioic acid, O,O-diethyl O(and S)-[2-(ethylthio)ethyl] esters
(paralysis and stimulation by)
IT 56-38-2, Parathion
(intestinal stimulation and paralysis by)
IT 8065-48-3, Systox
(paralyzing and stimulating effects on intestine)
L9 ANSWER 413 OF 434 CAPLUS COPYRIGHT 2003 ACS
AN 1965:38853 CAPLUS
DN 62:38853
OREF 62:6881e-h
TI The histochemistry of the **esterase** of mast cells.
AU Keller, R.
CS Dermatol. Universitatsklin., Zurich, Switz.
SO Schweizerische Medizinische Wochenschrift (1963), 93, 1504-5
CODEN: SMWOAS; ISSN: 0036-7672
DT Journal
LA German
CC 65 (Mammalian Biochemistry)
AB The influence of a number of inhibitor, on the **esterase**, leucine aminopeptidase, and ATPase of isolated rat mast cells was investigated. The compds. tested were: eserine (I), diisopropyl phosphorofluoridate (II), **Paraoxon** (III), Coroxon (IV), armine (V), Mipafox (VI), TEPP (VII), MgCl₂, CdCl₂, MnCl₂, CoCl₂, NiCl₂, CuCl₂, ZnCl₂, HgCl₂, Pb (NO₃)₂, AgNO₃, 1-fluoro-2,4-dinitrobenzene(VIII), formaldehyde (IX), phenylisocyanate (X), acetic anhydride (XI), ninhydrin (XII), iodoacetic acid (XIII), iodobenzoate (XIV), N-ethylmaleimide (XV), Na arsenite (XVI), Na arsenate (XVII), p-chloromercuribenzoate (XVIII), phenylmercurichloride (XIX), phenylacetic acid (XX), 2,4-dinitrophenol (XXI), NaN₃, NaCN, NaF, Na₂SO₃, phenol, EDTA, hydroxycinnamic acid (XXII), nicotinic acid amide (XXIII), and Na taurocholate (XXIV). The following compds. inhibited the activity of the **esterase** to about 25%: V, VI, MgCl₂, NiCl₂, CuCl₂, ZnCl₂, XVI, XVII, XX, and EDTA; to about 50%: II, III, IV, CdCl₂, CoCl₂, XIII, phenol, and XXII; to 75%: Na₂SO₃; completely (100%): HgCl₂, NaCN, and XXII. The activity was enhanced by XVIII, XIX, and NaF (25%).

The following compds. inhibited leucine aminopeptidase to about 25%: Pb(NO₃)₂, AgNO₃, VIII, IX, X, XIII, and XXIII; to about 50%; CoCl₂, NiCl₂, HgCl₂, XVII, XX, phenol, and XXIV; to 75%; CuCl₂, XI, XIX, XXI, and EDTA; completely: NaN₃, NaCN, Na₂SO₃. The following compds. inhibited the activity of ATPase to 25%: MgCl₂, NiCl₂, AgNO₃, IX, XX, and XXIV; to 50%; CaCl₂, VIII, and XXIII; to 75%: XI and NaCN; completely: XVI, XVII, Na₂SO₃, and EDTA. The results support the assumption that the nonspecific **esterase** is a chymotrypsin-like enzyme.

IT Mast cells
(esterases in, inhibitor effect on)

IT Cinnamic acid, hydroxy-
Phosphoric acid, diethyl p-nitrophenyl ester
Sodium arsenite
(effect on adenosinetriphosphatase, **esterase** and leucine aminopeptidase of mast cells)

IT Et₄P₂O₇
(effect on adenosinetriphosphatase, esters and leucine aminopeptidase of mast cells)

IT Nicotinamide, adenosinetriphosphatase
(**esterase** and leucine aminopeptidase in mast cells in response to)

IT Sodium fluoride, adenosinetriphosphatase
(**esterase** and leucine aminopeptidase of mast cells in presence of)

IT Benzoic acid, p-(chloromercuri)-, adenosinetriphosphatase
Lead nitrate, adenosinetriphosphatase
Magnesium chloride, adenosinetriphosphatase
Physostigmine, adenosinetriphosphatase
Silver nitrate, adenosinetriphosphatase
Taurocholic acid, sodium salt, adenosinetriphosphatase
(**esterase** and leucine aminopeptidase of mast cells in relation to)

IT Sodium azide, adenosinetriphosphatase
(**esterase** and leucine aminopeptidase of most cells in relation to)

IT Acetic acid, phenyl-, adenosinetriphosphatase
(**esterase** and leucine aminopeptidase of most cells response to)

IT 1,2,3-Indantrione, monohydrate (Ninhydrin), adenosinetriphosphatase
(esters and leucine aminopeptidase of mast cells in relation to)

IT Esterases
(in mast cells, inhibitor effect on)

IT Leucine aminopeptidase, L-Leucine aminoexopeptidase or Leucyl peptidase
(of mast cells)

IT 50-00-0, Formaldehyde 7447-39-4, Copper chloride, CuCl₂ 7487-94-7,
Mercury chloride, HgCl₂ 10108-64-2, Cadmium chloride
(adenosinetriphosphatase, **esterase** and leucine aminopeptidase of mast cells in relation to)

IT 7646-79-9, Cobalt chloride, CoCl₂
(adenosinetriphosphatase, **esterase** and leucine aminopeptidase of mast cells in response to)

IT 64-69-7, Acetic acid, iodo-
(adenosinetriphosphatase, **esterase** and leucine aminopeptidase of most cells in relation to)

IT 51-28-5, Phenol, 2,4-dinitro- 55-91-4, Isopropyl phosphorofluoridate, (C₃H₇O)₂PF₃ 60-00-4, Acetic acid, (ethylenedinitrilo)tetra- 70-34-8, Benzene, 1-fluoro-2,4-dinitro- 103-71-9, Isocyanic acid, phenyl ester 108-24-7, Acetic anhydride 143-33-9, Sodium cyanide 321-54-0, Coumarin, 3-chloro-7-hydroxy-4-methyl-, diethyl phosphate 371-86-8, Phosphorodiamidic fluoride, N,N'-diisopropyl- 546-71-4, Phosphonic acid, ethyl-, ethyl p-nitrophenyl ester 7631-89-2, Sodium arsenate 7718-54-9, Nickel chloride, NiCl₂ 7757-83-7, Sodium sulfite, Na₂SO₃

(effect on adenosinetriphosphatase, **esterase** and leucine aminopeptidase of mast cells)
IT 100-56-1, Mercury, chlorophenyl- 128-53-0, Maleimide, N-ethyl-
27323-35-9, Benzoic acid, iodoso-
(effect on adenosinetriphosphatase, esters and leucine aminopeptidase of mast cells)
IT 7773-01-5, Manganese chloride, MnCl₂
(mast cell adenosinetriphosphatase **esterase** and leucine aminopeptidase in relation to)
IT 7646-85-7, Zinc chloride
(mast cell enzyme response to)
IT 9000-83-3, Adenosinetriphosphatase
(of mast cells)

=> d his

(FILE 'HOME' ENTERED AT 08:30:09 ON 02 JUN 2003)

FILE 'CAPLUS' ENTERED AT 08:30:33 ON 02 JUN 2003

L1 882 S STAVUDINE
E ESTERASE
L2 28490 S E3
L3 5 S L1 AND L2
E PHYSOSTIGMINE
L4 6222 S E3-E7
L5 274 S L2 AND L4
L6 0 S L5 AND VIRAL
L7 1 S L1 AND L5
E PARAOXON
L8 2427 S E3
L9 434 S L8 AND L2

=>

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	174.63	174.84
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-15.62	-15.62

STN INTERNATIONAL LOGOFF AT 09:24:11 ON 02 JUN 2003

AN 1975:164856 CAPLUS
 DN 82:164856
 TI Effect upon drug toxicity of surgical interference with hepatic or renal function
 AU Selye, H.; Mecs, I.
 CS Inst. Medecine Chir. Exp., Univ. Montreal, Montreal, QC, Can.
 SO Acta Hepato-Gastroenterologica (1974), 21(3), 191-202; (4), 266-73
 CODEN: AHGSBY; ISSN: 0300-970X
 DT Journal
 LA English
 CC 1-5 (Pharmacodynamics)
 Section cross-reference(s): 2, 4, 13
 GI For diagram(s), see printed CA Issue.
 AB The effect of choledochus ligature, partial hepatectomy, partial nephrectomy, and the steroids, pregnenolone-16.alpha.-carbonitrile (PCN) [1434-54-4] and triamcinolone [124-94-7] on the toxicity of 175 drugs was detd. in rats. For example, the toxicity of glutethimide (I) [77-21-4] was inhibited by PCN and triamcinolone and increased by choledochus ligature, partial hepatectomy, and to a lesser extent, partial nephrectomy, whereas indomethacin [53-86-1] was detoxified by choledochus ligature and PCN but was unaffected by the other treatments. The toxicity of 77 compds. was decreased by PCN, but was potentiated by partial hepatectomy in only 53 of them. Triamcinolone inhibited the toxicity of 33 compds.
 ST drug toxicity liver kidney surgery; triamcinolone drug toxicity; pregnenolonecarbonitrile drug toxicity
 IT Detoxication
 (of pharmaceuticals)
 IT Kidney, metabolism
 Liver, metabolism
 (pharmaceutical detoxication by)
 IT 124-94-7 1434-54-4
 RL: BIOL (Biological study)
 (pharmaceuticals detoxication response to)
 IT 50-09-9 50-12-4 50-29-3, biological studies 51-12-7 51-28-5,
 biological studies 51-42-3 51-52-5 51-56-9 52-31-3 52-86-8
 53-21-4 53-86-1 54-11-5 54-21-7 54-64-8 54-95-5 55-65-2
 55-86-7 55-91-4 56-34-8 56-38-2 56-81-5, biological studies
 56-89-3, biological studies 57-06-7 57-30-7 57-44-3 57-53-4
 57-83-0, biological studies 57-94-3 58-15-1 58-25-3 58-73-1
 59-41-6 59-47-2 59-52-9 60-19-5 61-80-3 62-55-5 62-74-8
 62-75-9 64-17-5, biological studies 64-31-3 64-47-1 64-86-8
 66-76-2 66-81-9 67-21-0 67-66-3, biological studies 67-68-5,
 biological studies 67-96-9 71-27-2 71-63-6 71-73-8 71-78-3
 73-24-5, biological studies 75-15-0, biological studies 75-80-9
 76-44-8 77-21-4 78-34-2 78-44-4 79-06-1, biological studies
 81-23-2 81-25-4 81-81-2 82-58-6 83-44-3 83-67-0 86-50-0
 87-66-1 89-82-7 94-59-7 95-25-0 97-44-9 98-50-0 100-17-4
 100-61-8 103-72-0 103-84-4 103-90-2 107-07-3, biological studies
 107-12-0 107-13-1, biological studies 107-21-1, biological studies
 108-86-1 109-75-1 110-89-4, biological studies 114-49-8 118-74-1
 118-96-7 121-59-5 123-31-9, biological studies 124-87-8 125-30-4
 125-64-4 125-84-8 125-85-9 126-07-8 127-48-0 127-85-5 128-37-0
 129-46-4 131-73-7 132-60-5 134-72-5 137-58-6 145-41-5 145-42-6
 146-56-5 151-67-7 152-16-9 154-42-7 156-57-0 288-13-1 298-46-4
 299-78-5 300-62-9 300-68-5 302-95-4 306-07-0 315-30-0 316-42-7
 318-98-9 361-09-1 434-13-9 466-06-8 492-18-2 496-72-0 513-10-0
 530-78-9 551-06-4 553-69-5 554-13-2 563-12-2 584-84-9 593-74-8
 630-93-3 830-89-7 863-57-0 1095-90-5 1229-29-4 1303-28-2
 1401-55-4 1421-86-9 1553-60-2 1639-60-7 1772-03-8 1867-66-9
 2104-64-5 2181-04-6 3238-60-6 3820-67-5 4044-65-9 5341-61-7
 5907-38-0 7447-39-4, biological studies 7487-94-7 7601-89-0

7723-14-0, biological studies 7785-87-7 7790-86-5 9011-04-5
10025-82-8 10099-58-8 10108-64-2 10138-52-0 13410-01-0
15256-58-3 15500-66-0 15571-91-2 15687-27-1 18911-13-2
39377-61-2 55347-53-0

RL: PRP (Properties)

(toxicity of, kidney and liver and steroids effect on)

=>

AN 1975:164856 CAPLUS
 DN 82:164856
 TI Effect upon drug toxicity of surgical interference with hepatic or renal function
 AU Selye, H.; Mecs, I.
 CS Inst. Medecine Chir. Exp., Univ. Montreal, Montreal, QC, Can.
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 CODEN: AHGSBY; ISSN: 0300-970X
 DT Journal
 LA English
 CC 1-5 (Pharmacodynamics)
 Section cross-reference(s): 2, 4, 13
 GI For diagram(s), see printed CA Issue.
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 ST drug toxicity liver kidney surgery; triamcinolone drug toxicity; pregnenolonecarbonitrile drug toxicity
 IT Detoxication
 (of pharmaceuticals)
 IT Kidney, metabolism
 Liver, metabolism
 (pharmaceutical detoxication by)
 IT 124-94-7 1434-54-4
 RL: BIOL (Biological study)
 (pharmaceuticals detoxication response to)
 IT 50-09-9 50-12-4 50-29-3, biological studies 51-12-7 51-28-5,
 biological studies 51-42-3 51-52-5 51-56-9 52-31-3 52-86-8
 53-21-4 53-86-1 54-11-5 54-21-7 54-64-8 54-95-5 55-65-2
 55-86-7 55-91-4 56-34-8 56-38-2 56-81-5, biological studies
 56-89-3, biological studies 57-06-7 57-30-7 57-44-3 57-53-4
 57-83-0, biological studies 57-94-3 58-15-1 58-25-3 58-73-1
 59-41-6 59-47-2 59-52-9 60-19-5 61-80-3 62-55-5 62-74-8
 62-75-9 64-17-5, biological studies 64-31-3 64-47-1 64-86-8
 66-76-2 66-81-9 67-21-0 67-66-3, biological studies 67-68-5,
 biological studies 67-96-9 71-27-2 71-63-6 71-73-8 71-78-3
 73-24-5, biological studies 75-15-0, biological studies 75-80-9
 76-44-8 77-21-4 78-34-2 78-44-4 79-06-1, biological studies
 81-23-2 81-25-4 81-81-2 82-58-6 83-44-3 83-67-0 86-50-0
 87-66-1 89-82-7 94-59-7 95-25-0 97-44-9 98-50-0 100-17-4
 100-61-8 103-72-0 103-84-4 103-90-2 107-07-3, biological studies
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 129-46-4 131-73-7 132-60-5 134-72-5 137-58-6 145-41-5 145-42-6
 146-56-5 151-67-7 152-16-9 154-42-7 156-57-0 288-13-1 298-46-4
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 318-98-9 361-09-1 434-13-9 466-06-8 492-18-2 496-72-0 513-10-0
 530-78-9 551-06-4 553-69-5 554-13-2 563-12-2 584-84-9 593-74-8
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39377-61-2 55347-53-0

RL: PRP (Properties)

(toxicity of, kidney and liver and steroids effect on)

=>

DN 66:17758
TI Protective effect of aldrin against the toxicity of organophosphate anticholinesterases
AU Triolo, Anthony J.; Coon, Julius M.
CS Jefferson Med. Coll., Philadelphia, PA, USA
SO Journal of Pharmacology and Experimental Therapeutics (1966), 154(3), 613-23
CODEN: JPETAB; ISSN: 0022-3565
DT Journal
LA German/French
CC 14 (Toxicology)
AB A single oral dose of 16 mg. of aldrin/kg. protected mice 4 days later against parathion, para-oxon, tetraethyl pyrophosphate, diisopropyl fluorophosphate, O-ethyl O-(p-nitrophenyl) phosphorothioate, Guthion, tri-o-tolyl phosphate, and physostigmine, but not against octamethylpyrophosphoramid (OMPA) or neostigmine. One hour after aldrin, the toxicity of parathion was increased, whereas, from 16 hrs. to 12 days after aldrin, animals were significantly protected. This effect of aldrin reached its max. in .apprx.4 days, and 1 mg./kg. provided significant protection. Two days after aldrin, A-esterase, which detoxifies para-oxon, increased 38% in the liver but decreased 50% in the plasma, and plasma B-esterase, which is inhibited by para-oxon, was increased 24%. Aldrin had no effect on the inhibitory action of para-oxon on plasma cholinesterase, but it reduced this action of para-oxon in the brain. This is in accord with the finding that aldrin failed to protect against OMPA or neostigmine, which differ from the other anticholinesterases tested in being poor in vivo inhibitors of brain cholinesterase. Ethionine abolished the protective effect of aldrin against the toxicity and brain cholinesterase-inhibiting action of para-oxon and prevented the aldrin-induced increases in plasma B-esterase and liver A-esterase. Ethionine, alone, however, increased the mortalities after parathion and para-oxon. Though the increases in A- and B-esterases would be expected to decrease the toxicities of parathion and para-oxon, other factors possibly involving the central nervous system may play a role in the protective effect of aldrin against organophosphate poisoning.
ST ORGANOPHOSPHATES ALDRIN; ANTICHOINESTERASE ALDRIN; ALDRIN PESTICIDES; PESTICIDES ALDRIN; PESTICIDES ALDRIN; ALDRIN PESTICIDES; ANTICHOINESTERASE ALDRIN; ORGANOPHOSPHATES ALDRIN
IT Brain, composition
(cholinesterase inhibition by organophosphate in, ethionine inhibition of aldrin protection of)
IT Poisoning
(organophosphate; aldrin protection against)
IT 55-17-4
IT RL: BIOL (Biological study)
(aldrin protective action against p-oxon anticholinesterase action inhibition by)
IT 9013-79-0, Esterases
(in blood, brain and liver in organophosphate poisoning, aldrin effect on)
IT 9001-08-5, Esterases, choline
(inhibition of, by organophosphate in brain, aldrin protection of, ethionine antagonism to)
IT 309-00-2
IT RL: PROC (Process)
(organophosphate poisoning-protective action of)
IT 55-91-4 56-38-2 57-47-6 78-30-8 86-50-0 107-49-3 311-45-5
15576-30-4
IT RL: BIOL (Biological study)
(poisoning by, aldrin protection against)

DN 66:17758
TI Protective effect of aldrin against the toxicity of organophosphate anticholinesterases
AU Triolo, Anthony J.; Coon, Julius M.
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SO Journal of Pharmacology and Experimental Therapeutics (1966), 154(3), 613-23
CODEN: JPETAB; ISSN: 0022-3565
DT Journal
LA German/French
CC 14 (Toxicology)
AB A single oral dose of 16 mg. of aldrin/kg. protected mice 4 days later against parathion, para-oxon, tetraethyl pyrophosphate, diisopropyl fluorophosphate, O-ethyl O-(p-nitrophenyl) phosphorothioate, Guthion, tri-o-tolyl phosphate, and physostigmine, but not against octamethylpyrophosphoramide (OMPA) or neostigmine. One hour after aldrin, the toxicity of parathion was increased, whereas, from 16 hrs. to 12 days after aldrin, animals were significantly protected. This effect of aldrin reached its max. in .apprx.4 days, and 1 mg./kg. provided significant protection. Two days after aldrin, A-esterase, which detoxifies para-oxon, increased 38% in the liver but decreased 50% in the plasma, and plasma B-esterase, which is inhibited by para-oxon, was increased 24%. Aldrin had no effect on the inhibitory action of para-oxon on plasma cholinesterase, but it reduced this action of para-oxon in the brain. This is in accord with the finding that aldrin failed to protect against OMPA or neostigmine, which differ from the other anticholinesterases tested in being poor in vivo inhibitors of brain cholinesterase. Ethionine abolished the protective effect of aldrin against the toxicity and brain cholinesterase-inhibiting action of para-oxon and prevented the aldrin-induced increases in plasma B-esterase and liver A-esterase. Ethionine, alone, however, increased the mortalities after parathion and para-oxon. Though the increases in A- and B-esterases would be expected to decrease the toxicities of parathion and para-oxon, other factors possibly involving the central nervous system may play a role in the protective effect of aldrin against organophosphate poisoning.
ST ORGANOPHOSPHATES ALDRIN; ANTICHOLINESTERASE ALDRIN; ALDRIN PESTICIDES; PESTICIDES ALDRIN; PESTICIDES ALDRIN; ALDRIN PESTICIDES; ANTICHOLINESTERASE ALDRIN; ORGANOPHOSPHATES ALDRIN
IT Brain, composition
(cholinesterase inhibition by organophosphate in, ethionine inhibition of aldrin protection of)
IT Poisoning
(organophosphate, aldrin protection against)
IT 55-17-4
RL: BIOL (Biological study)
(aldrin protective action against p-oxon anticholinesterase action inhibition by)
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(in blood, brain and liver in organophosphate poisoning, aldrin effect on)
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RL: PROC (Process)
(organophosphate poisoning-protective action of)
IT 55-91-4 56-38-2 57-47-6 78-30-8 ,86-50-0 107-49-3 311-45-5
15576-30-4
RL: BIOL (Biological study)
(poisoning by, aldrin protection against).

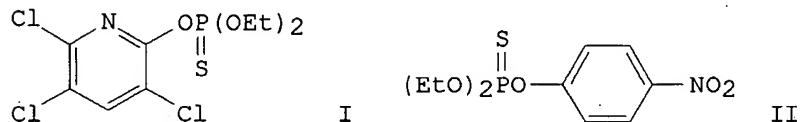
DN 103:66085
TI Interethnic differences of human serum paraoxonase activity-relevance for the detoxification of organophosphorous compounds
AU Geldmacher-Von Mallinckrodt, M.; Diepgen, T. L.; Enders, P. W.
CS Inst. Rechtsmed., Univ. Erlangen-Nuernberg, Erlangen, D-8520, Fed. Rep. Ger.
SO Archives Belges de Medecine Sociale, Hygiene, Medecine du Travail et Medecine Legale (1984), Suppl.(Proc.-World Congr. "New Compd. Biol. Chem. Warf.: Toxicol. Eval., 1st, 1984), 243-51
CODEN: ABMHAM; ISSN: 0003-9578
DT Journal; General Review
LA English
CC 4-0 (Toxicology)
AB A review with 19 refs. on interethnic differences of human serum paraoxonase [117698-12-1] activity and its relevance for the detoxication of organophosphorus compds., i.e., paraoxon [311-45-5].
ST review serum paraoxonase human genetics; detoxication organophosphate serum paraoxonase review
IT Detoxication
 (of organophosphorus compds., interethnic differences of human blood serum paraoxonase in relation to)
IT Genetics
 (paraoxonase of human blood serum in relation to)
IT Blood serum
 (paraoxonase of, of humans, interethnic differences of, detoxication of organophosphorus compds. in relation to)
IT 311-45-5 7723-14-0D, org: compds.
 RL: BIOL (Biological study)
 (detoxication of, interethnic differences of human blood serum paraoxonase in relation to)
IT 117698-12-1
 RL: BIOL (Biological study)
 (of blood serum, of humans, interethnic differences of, detoxication of organophosphorus compds. in relation to)

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 (of organophosphorus compds., interethnic differences of human blood serum paraoxonase in relation to)
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 (paraoxonase of, of humans, interethnic differences of, detoxication of organophosphorus compds. in relation to)
IT 311-45-5 7723-14-0D, org. compds.
 RL: BIOL (Biological study)
 (detoxication of, interethnic differences of human blood serum paraoxonase in relation to)
IT 117698-12-1
 RL: BIOL (Biological study)
 (of blood serum, of humans, interethnic differences of, detoxication of organophosphorus compds. in relation to)

=>

DN 102:161862
TI Metabolic activation of phosphorothioate pesticides: role of the liver
AU Sultatos, Lester G.; Minor, Lerna D.; Murphy, Sheldon D.
CS Med. Cent., Louisiana State Univ., New Orleans, LA, USA
SO Journal of Pharmacology and Experimental Therapeutics (1985), 232(3),
624-8
CODEN: JPETAB; ISSN: 0022-3565
DT Journal
LA English
CC 4-4 (Toxicology)
GI



AB Mouse liver perfusion studies *in situ* revealed that the cholinesterase inhibitor chlorpyrifos oxon [5598-15-2] produced by the liver from the phosphorothioate pesticide chlorpyrifos (I) [2921-88-2] was quickly detoxified within the liver, thereby preventing its exit from the liver in the effluent. In contrast, when the pesticide parathion (II) [56-38-2] was perfused as a substrate, a substantial amt. of the toxic metabolite paraoxon [311-45-5] was found in exiting perfusate. Pesticide concns. (5-15 .mu.M) used in the perfusion studies *in situ* were similar to their hepatic portal blood concns. *in vivo* (2.32-12.95 .mu.M) after i.p. administration of lethal or near LDs. Moreover, the half-life for elimination of paraoxon by mouse blood *in vitro* was 8.6 min, a rate sufficiently low to allow passage of paraoxon to extrahepatic target tissues from liver *in vivo*. Apparently, in the mouse, the acute toxicity of chlorpyrifos is mediated by extrahepatic prodn. of oxon, whereas parathion is likely mediated by hepatic and extrahepatic activation.

ST liver chlorpyrifos parathion metab

IT Liver, metabolism

(chlorpyrifos and parathion metabolic activation in, perfusion in relation to)

IT Blood

(chlorpyrifos and parathion of, after administration, liver in relation to)

IT 311-45-5 5598-15-2

RL: FORM (Formation, nonpreparative)

(formation of, by liver, perfusion in relation to)

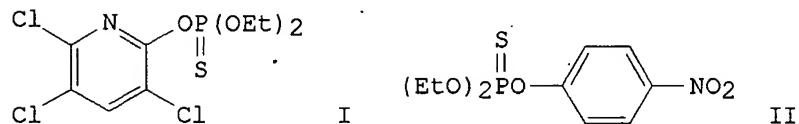
IT 56-38-2 2921-88-2

RL: BIOL (Biological study)

(metabolic activation of, liver perfusion in relation to)

=>

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ST liver chlorpyrifos parathion metab

IT Liver, metabolism

(chlorpyrifos and parathion metabolic activation in, perfusion in relation to)

IT Blood

(chlorpyrifos and parathion of, after administration, liver in relation to)

IT 311-45-5 5598-15-2

RL: FORM (Formation, nonpreparative)

(formation of, by liver, perfusion in relation to)

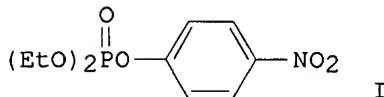
IT 56-38-2 2921-88-2

RL: BIOL (Biological study)

(metabolic activation of, liver perfusion in relation to)

=>

DN 98:66771
TI Enzymic detoxication of organophosphorus insecticides and nerve gases in primates
AU Losch, H.; Losch, K.; Haselmeyer, K. H.; Chemnitius, J. M.; Zech, R.
CS Zent. Biochem., Georg-August-Univ., Goettingen, 3400, Fed. Rep. Ger.
SO Arzneimittel-Forschung (1982), 32(12), 1523-9
CODEN: ARZNAD; ISSN: 0004-4172
DT Journal
LA German
CC 4-4 (Toxicology)
GI



AB The detoxication of organophosphorus compds. by phosphorylphosphatases was studied in primates. Taking into account the distribution of paraoxonase and DFPase (EC 3.8.2.1) [9032-18-2] in different tissues of the monkey (Macaca mulatta), the total detoxicating capacity for paraoxon (I) [311-45-5] and diiso-Pr phosphorofluoridate (DFP) [55-91-4] was detd. acetylcholinesterase (EC 3.1.1.7) (AChE) [9000-81-1] of human brain was inhibited in vitro by I and DFP. By using the rate consts. of AChE inhibition and synthesis, the concns. of organophosphorus inhibitors were calcd., which would reduce the steady-state AChE activity to 20% of normal. This acute ineffective concn. is 7.6 .times. 10-8 g/kg for DFP and 2.3 .times. 10-8 g/kg for I. From substrate kinetics of the phosphorylphosphatases, the time course of I and DFP detoxication in primates could be calcd. The time needed by phosphorylphosphatases to reduce a certain dose of an organophosphorus compd. to the acute ineffective concn. is referred to as effective detoxication time. The effective detoxication time was detd. for different concns. of I and DFP and compared with the time needed by the organophosphate concns. to inhibit AChE activity to 12.5% of normal. The significance of in vitro data for the evaluation of dose limits of organophosphate toxicity in vivo is discussed.

ST paraoxon enzyme detoxification; phosphorofluoridate enzyme detoxification; enzyme detoxification org phosphate

IT Brain

Kidney

Liver

Lung

Muscle

Organ

Spleen

(diisopropyl phosphorofluoridate and paraoxon detoxification by, in humans)

IT Kinetics, enzymic

(of acetylcholinesterase and diisopropyl fluorophosphatase and paraoxonase detoxification of org. phosphorus compds.)

IT Blood serum

(paraoxon detoxification by, in humans)

IT 55-91-4 311-45-5

RL: BIOL (Biological study)

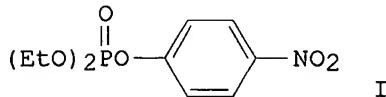
(detoxification of, kinetics of)

IT 9000-81-1

RL: BIOL (Biological study)

(diisopropyl phosphorofluoridate and paraoxon inhibition of,

DN 98:66771
TI Enzymic detoxication of organophosphorus insecticides and nerve gases in primates
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CS Zent. Biochem., Georg-August-Univ., Goettingen, 3400, Fed. Rep. Ger.
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Liver
Lung
Muscle
Organ
Spleen

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IT Kinetics, enzymic
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IT Blood serum
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IT 55-91-4 311-45-5
RL: BIOL (Biological study)
(detoxification of, kinetics of)

IT 9000-81-1
RL: BIOL (Biological study)
(diisopropyl phosphorofluoridate and paraoxon inhibition of,

detoxification kinetics in relation to)

IT 9032-18-2

RL: PROC (Process)

(diisopropyl phosphorofluoridate inhibition of, detoxification kinetics
in relation to)

IT 117698-12-1

RL: PROC (Process)

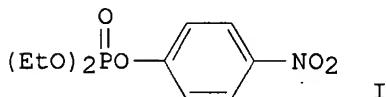
(paraoxon inhibition of, detoxification kinetics in relation to)

=>

detoxification kinetics in relation to)
IT 9032-18-2
RL: PROC (Process)
(diisopropyl phosphorofluoridate inhibition of, detoxification kinetics
in relation to)
IT 117698-12-1
RL: PROC (Process)
(paraoxon inhibition of, detoxification kinetics in relation to)

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AU Losch, H.; Losch, K.; Haselmeyer, K. H.; Chemnitius, J. M.; Zech, R.
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CODEN: ARZNAD; ISSN: 0004-4172
DT Journal
LA German
CC 4-4 (Toxicology)
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IT Kinetics, enzymic

(of acetylcholinesterase and diisopropyl fluorophosphatase and paraoxonase detoxification of org. phosphorus compds.)

IT Blood serum

(paraoxon detoxification by, in humans)

IT 55-91-4 311-45-5

IT RL: BIOL (Biological study)

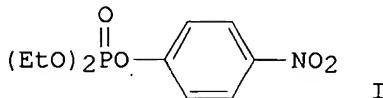
(detoxification of, kinetics of)

IT 9000-81-1

IT RL: BIOL (Biological study)

(diisopropyl phosphorofluoridate and paraoxon inhibition of,

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Kidney
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Lung
Muscle
Organ
Spleen

(diisopropyl phosphorofluoridate and paraoxon detoxification by, in humans)

IT Kinetics, enzymic
(of acetylcholinesterase and diisopropyl fluorophosphatase and paraoxonase detoxification of org. phosphorus compds.)

IT Blood serum
(paraoxon detoxification by, in humans)

IT 55-91-4 311-45-5
RL: BIOL (Biological study)
(detoxification of, kinetics of)

IT 9000-81-1
RL: BIOL (Biological study)
(diisopropyl phosphorofluoridate and paraoxon inhibition of,

detoxification kinetics in relation to)
IT 9032-18-2
RL: PROC (Process)
(diisopropyl phosphorofluoridate inhibition of, detoxification kinetics
in relation to)
IT 117698-12-1
RL: PROC (Process)
(paraoxon inhibition of, detoxification kinetics in relation to)

=>

detoxification kinetics in relation to)
IT 9032-18-2
RL: PROC (Process)
(diisopropyl phosphorofluoridate inhibition of, detoxification kinetics
in relation to)
IT 117698-12-1
RL: PROC (Process)
(paraoxon inhibition of, detoxification kinetics in relation to)

=>

DN 100:46971
TI Synthesis and biological activity studies of selected organophosphorus esters
AU McElroy, Roger D.; Chambers, Howard W.
CS Dep. Entomol., Mississippi State Univ., Mississippi State, MS, 39762, USA
SO Journal of Agricultural and Food Chemistry (1984), 32(1), 119-23
CODEN: JAFCAU; ISSN: 0021-8561
DT Journal
LA English
CC 5-4 (Agrochemical Bioregulators)
AB Thirty organophosphorus esters (structurally similar DEF analogs) were synthesized and evaluated as possible insecticide (methyl paraoxon [950-35-6]) synergists against boll weevils, *Anthonomus grandis*. B-esterase [9016-18-6] and acetylcholinesterase activity from organophosphorus-susceptible weevils were measured spectrophotometrically with S-Ph thiobenzoate and acetylthiocholine as substrates. The structure-biol. activity relation may be divided into 3 major effects, i.e., a lipophilic effect, an electronic effect, and a steric effect. In vitro and in vivo inhibition and toxicity data support the hypothesis that synergism of Me paraoxon results from the inhibition of the esterase hydrolyzing S-Ph thiobenzoate by selected organophosphorus esters.
ST insecticide synergist boll weevil methyl paraoxon; phosphorotriithioate insecticide synergist
IT Insecticides
 (esterase-inhibiting, synergists for, tributylphosphorotriithioate analogs as)
IT *Anthonomus grandis*
 (insecticide synergists against, tributylphosphorotriithioate analogs as)
IT Molecular structure-biological activity relationship
 (insecticidal synergistic, of tributylphosphorotriithioate analogs)
IT 9016-18-6
RL: PROC (Process)
 (inhibition of, by insecticide, organophosphorus ester synergists in)
IT 78-48-8P 1642-44-0P 2797-64-0P 3819-72-5P 4081-23-6P 24067-01-4P
24067-02-5P 26115-85-5P 26115-86-6P 30299-04-8P 68598-35-6P
68598-36-7P 68598-37-8P 68598-38-9P 68598-39-0P 68598-40-3P
68598-41-4P 68598-42-5P 78788-15-5P 85480-01-9P 85480-02-0P
85480-03-1P 85480-04-2P 85480-05-3P 85480-06-4P 85480-07-5P
85480-08-6P 85480-09-7P 85480-10-0P 85480-11-1P
RL: SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and insecticide synergistic activity of, against boll weevil)
IT 950-35-6
RL: BIOL (Biological study)
 (tridecylphosphorotriithioate analogs as insecticidal synergist of, against bollweevils)

=>

DN 100:46971
TI Synthesis and biological activity studies of selected organophosphorus esters
AU McElroy, Roger D.; Chambers, Howard W.
CS Dep. Entomol., Mississippi State Univ., Mississippi State, MS, 39762, USA
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ST insecticide synergist boll weevil methyl paraoxon; phosphorotriithioate insecticide synergist
IT Insecticides
 (esterase-inhibiting, synergists for, tributylphosphorotriithioate analogs as)
IT *Anthonomus grandis*
 (insecticide synergists against, tributylphosphorotriithioate analogs as)
IT Molecular structure-biological activity relationship
 (insecticidal synergistic, of tributylphosphorotriithioate analogs)
IT 9016-18-6
RL: PROC (Process)
 (inhibition of, by insecticide, organophosphorus ester synergists in)
IT 78-48-8P 1642-44-0P 2797-64-0P 3819-72-5P 4081-23-6P 24067-01-4P
24067-02-5P 26115-85-5P 26115-86-6P 30299-04-8P 68598-35-6P
68598-36-7P 68598-37-8P 68598-38-9P 68598-39-0P 68598-40-3P
68598-41-4P 68598-42-5P 78788-15-5P 85480-01-9P 85480-02-0P
85480-03-1P 85480-04-2P 85480-05-3P 85480-06-4P 85480-07-5P
85480-08-6P 85480-09-7P 85480-10-0P 85480-11-1P
RL: SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and insecticide synergistic activity of, against boll weevil)
IT 950-35-6
RL: BIOL (Biological study)
 (tridecylphosphorotriithioate analogs as insecticidal synergist of, against bollweevils)

=>

DN 130:177179
TI Synthesis, anti-human immunodeficiency virus activity and **esterase** lability of some novel carboxylic ester-modified phosphoramidate derivatives of **stavudine** (d4T)
AU McGuigan, C.; Sutton, P. W.; Cahard, D.; Turner, K.; O'Leary, G.; Wang, Y.; Gumbleton, M.; De Clercq, E.; Balzarini, J.
CS Welsh School Pharmacy, Cardiff University, Cardiff, CF1 3XF, UK
SO Antiviral Chemistry & Chemotherapy (1998), 9(6), 473-479
CODEN: ACCHEH; ISSN: 0956-3202
PB International Medical Press
DT Journal
LA English
CC 1-5 (Pharmacology)
Section cross-reference(s): 33
AB We report the design, synthesis and antiviral evaluation of a series of lipophilic, masked phosphoramidate derivs. of the anti-human immunodeficiency virus (HIV) nucleoside analog d4T, designed to act as membrane-sol. pro-drug forms for the free nucleotide. In particular, we report a series of 12 novel compds. with systematic variation in the structure of the carboxylate ester function. In order to rationalize the changes in antiviral action with variation of this moiety we applied the recently developed 31P NMR-based assay for carboxyesterase lability to this series. However, no clear pos. correlation emerged, indicating that, at least within this series, factors other than simple **esterase** lability may be the major determinants of antiviral potency.
ST virus immunodeficiency human phosphoramidate deriv **stavudine** prepn; HIV virus phosphoramidate deriv **stavudine** prepn
IT Antiviral agents
Human immunodeficiency virus 1
(prepn. and anti-HIV virucidal activity and **esterase** lability of carboxylic ester-modified phosphoramidate derivs. of **stavudine**)
IT 9016-18-6, **Esterase**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(pig liver; prepn. and anti-HIV virucidal activity and **esterase** lability of carboxylic ester-modified phosphoramidate derivs. of **stavudine**)
IT 3056-17-5DP, **Stavudine**, derivs. 173070-83-2P 178469-24-4P
184031-34-3P 184031-40-1P 184031-42-3P 220592-56-3P 220592-57-4P
220592-58-5P 220592-59-6P 220592-60-9P 220592-61-0P 220592-62-1P
220592-74-5P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(prepn. and anti-HIV virucidal activity and **esterase** lability of carboxylic ester-modified phosphoramidate derivs. of **stavudine**)
IT 142629-80-9 183370-70-9 220592-63-2 220592-64-3 220592-65-4
220592-66-5 220592-67-6 220592-68-7 220592-69-8 220592-70-1
220592-71-2 220592-72-3 220592-73-4
RL: RCT (Reactant); RACT (Reactant or reagent)
(prepn. and anti-HIV virucidal activity and **esterase** lability of carboxylic ester-modified phosphoramidate derivs. of **stavudine**)
IT 180076-92-0P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. and anti-HIV virucidal activity and **esterase** lability of carboxylic ester-modified phosphoramidate derivs. of **stavudine**)
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE

- (1) Balzarini, J; Molecular Pharmacology 1996, V50, P1207 CAPLUS
- (2) Balzarini, J; Proceedings of the National Academy of Sciences USA 1996, V93, P7295 CAPLUS
- (3) McGuigan, C; Antiviral Chemistry and Chemotherapy 1998, V9, P109 CAPLUS
- (4) McGuigan, C; Journal of Medicinal Chemistry 1993, V36, P1048 CAPLUS
- (5) McGuigan, C; Journal of Medicinal Chemistry 1996, V39, P1748 CAPLUS
- (6) Meier, C; Synthesis Letters 1998, P233 CAPLUS

DN 128:289775
TI Synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivatives of d4T (**stavudine**): esterase hydrolysis as a rapid predictive test for antiviral potency
AU McGuigan, C.; Tsang, H.-W.; Sutton, P. W.; De Clercq, E.; Balzarini, J.
CS Welsh School Pharmacy, University Wales Cardiff, Cardiff, CF1 3XF, UK
SO Antiviral Chemistry & Chemotherapy (1998), 9(2), 109-115
CODEN: ACCHEH; ISSN: 0956-3202
PB International Medical Press
DT Journal
LA English
CC 1-5 (Pharmacology)
Section cross-reference(s): 7, 33
AB Novel chain-extended nucleoside phosphoramidates of the anti-human immunodeficiency virus (HIV) drug d4T (**stavudine**) have been prepd. as possible membrane-permeable prodrugs of the bio-active free 5'-monophosphates. Phosphorochloridate chem. gave the target compds. in moderate to high yields, and all materials were fully characterized by spectroscopic and anal. methods. The compds. are related to the previously reported Ph methoxyalaninyl deriv. of d4T, which was shown to be a potent and selective inhibitor of HIV. In this study the amino acid nitrogen and ester moieties were sep'd. by methylene spacers of between two and six carbon atoms. In vitro evaluation of these compds. indicated an almost complete lack of anti-HIV activity, the compds. being several orders of magnitude less potent than the corresponding .alpha.-amino acid derivs. The reasons for the virtual lack of anti-HIV activity appear to involve poor enzyme-mediated hydrolysis.
ST nucleoside phosphoramidate anti human immunodeficiency virus
IT Antiviral agents
Human immunodeficiency virus 1
Human immunodeficiency virus 2
(synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))
IT 9016-18-6, **Esterase**
RL: BPR (Biological process); BSU (Biological study, unclassified); CAT (Catalyst use); BIOL (Biological study); PROC (Process); USES (Uses)
(pig liver; synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))
IT 184031-47-8P 205991-44-2P 205991-51-1P 205991-52-2P 205991-53-3P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))
IT 205991-46-4P 205991-47-5P 205991-48-6P 205991-49-7P 205991-50-0P
RL: BPN (Biosynthetic preparation); BIOL (Biological study); PREP (Preparation)
(synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))
IT 770-12-7, Phenyl phosphorodichloridate 1926-80-3, 6-Aminocaproic acid methyl ester hydrochloride 3056-17-5, **Stavudine** 3196-73-4, .beta.-Alanine methyl ester hydrochloride 13031-60-2, 4-Aminobutanoic acid methyl ester hydrochloride 17994-94-4, 7-Aminoheptanoic acid methyl ester hydrochloride 29840-56-0, 5-Aminopentanoic acid methyl ester hydrochloride 173070-83-2 173070-84-3
RL: RCT (Reactant); RACT (Reactant or reagent)
(synthesis and anti-HIV activity of some novel chain-extended phosphoramidate derivs. of didehydrodeoxythymidine (d4T))
IT 205991-54-4P 205991-55-5P 205991-56-6P 205991-57-7P 205991-58-8P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)

(synthesis and anti-HIV activity of some novel chain-extended
phosphoramidate derivs. of didehydrodeoxythymidine (d4T))

DN 126:271667
TI A rational strategy for the design of anti-hepatitis B virus nucleotide derivatives
AU Perigaud, Christian; Gosselin, Gilles; Imbach, Jean-Louis
CS Laboratoire de Chimie Bioorganique, UMR CNRS 5625, Montpellier, 34095, Fr.
SO Antiviral Therapy (1996), 1(Suppl. 4, Therapies for Viral Hepatitis), 39-46
CODEN: ANTHFA; ISSN: 1359-6535
PB International Medical Press
DT Journal; General Review
LA English
CC 1-0 (Pharmacology)
AB A review with 42 refs. The potential in antiviral chemotherapy of a pronucleotide approach using mononucleoside phosphotriesters which incorporate S-acyl-2-thioethyl (SATE) groups as esterase-labile transient phosphate protectors is discussed in detail. The use of this approach leads to an increase in the antiretroviral activity of two well-established anti-HIV drugs, namely 2',3'-dideoxyadenosine (ddA) and 2',3'-didehydro-2',3'-dideoxythymidine (**stavudine** or d4T). Moreover, in the case of acyclovir, which is currently used as therapeutic agent for the treatment of herpes virus infections, the corresponding bis(SATE) pronucleotides have emerged as potent and selective inhibitors of the hepatitis B virus replication.
ST. review hepatitis virucide nucleotide deriv
IT Antiviral agents
Hepatitis B virus
(strategy for design of anti-hepatitis B virus nucleotide derivs.)
IT Nucleotides, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(strategy for design of anti-hepatitis B virus nucleotide derivs.)

=>

DN 30:856
OREF 30:124b-d
TI The **esterase** activity of human blood plasma
AU Vahlquist, Bo
SO Skand. Arch. Physiol. (1935), 72, 133-60
DT Journal
LA Unavailable
CC 11A (Biological Chemistry: General)
AB To decide whether human plasma contains a specific choline **esterase** or the hydrolysis is brought about by the ordinary lipase, a study was made by various methods. Cataphoretically both activities moved strictly parallel in the elec. field and independently of the migration of the albumin and globulin. Similarly quinine, atoxyl and **physostigmine** inhibited the action of the **esterase** no matter what substrate was employed (acetylcholine, tributyrin or Me butyrate). Parallel detns. of choline and tributyrin **esterase** activity were made on different individuals under a great variety of conditions. The correlation of all these results was so great that the correlation coeff. was 0.92 ± 0.02 . All 3 modes of attack on this problem indicate, therefore, that there is no specific acetylcholine **esterase**. The **esterase** content is not appreciably affected by ingestion of food, muscular exercise, nervous excitement, menstruation or pregnancy. Under conditions of abnormal muscular spasms such as bronchial asthma or ulcus ventriculi the values are relatively low but still within the normal range. Only in tuberculosis is the **esterase** content abnormally low. The **esterase** apparently can only act to protect the organism against an accumulation of acetylcholine in the blood.

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TI The histochemistry of the **esterase** of mast cells
AU Keller, R.
CS Dermatol. Universitatsklin., Zurich, Switz.
SO Schweizerische Medizinische Wochenschrift (1963), 93, 1504-5
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DT Journal
LA German
CC 65 (Mammalian Biochemistry)
AB The influence of a number of inhibitor, on the **esterase**, leucine aminopeptidase, and ATPase of isolated rat mast cells was investigated. The compds. tested were: eserine (I), diisopropyl phosphorofluoridate (II), **Paraoxon** (III), Coroxon (IV), armine (V), Mipafox (VI), TEPP (VII), MgCl₂, CdCl₂, MnCl₂, CoCl₂, NiCl₂, CuCl₂, ZnCl₂, HgCl₂, Pb (NO₃)₂, AgNO₃, 1-fluoro-2,4-dinitrobenzene(VIII), formaldehyde (IX), phenylisocyanate (X), acetic anhydride (XI), ninhydrin (XII), iodoacetic acid (XIII), iodobenzoate (XIV), N-ethylmaleimide (XV), Na arsenite (XVI), Na arsenate (XVII), p-chloromercuribenzoate (XVIII), phenylmercurichloride (XIX), phenylacetic acid (XX), 2,4-dinitrophenol (XXI), NaN₃, NaCN, NaF, Na₂SO₃, phenol, EDTA, hydroxycinnamic acid (XXII), nicotinic acid amide (XXIII), and Na taurocholate (XXIV). The following compds. inhibited the activity of the **esterase** to about 25%: V, VI, MgCl₂, NiCl₂, CuCl₂, ZnCl₂, XVI, XVII, XX, and EDTA; to about 50%: II, III, IV, CdCl₂, CoCl₂, XIII, phenol, and XXII; to 75%: Na₂SO₃; completely (100%): HgCl₂, NaCN, and XXII. The activity was enhanced by XVIII, XIX, and NaF (25%). The following compds. inhibited leucine aminopeptidase to about 25%: Pb(NO₃)₂, AgNO₃, VIII, IX, X, XIII, and XXIII; to about 50%: CoCl₂, NiCl₂, HgCl₂, XVII, XX, phenol, and XXIV; to 75%: CuCl₂, XI, XIX, XXI, and EDTA; completely: NaN₃, NaCN, Na₂SO₃. The following compds. inhibited the activity of ATPase to 25%: MgCl₂, NiCl₂, AgNO₃, IX, XX, and XXIV; to 50%: CaCl₂, VIII, and XXIII; to 75%: XI and NaCN; completely: XVI, XVII, Na₂SO₃, and EDTA. The results support the assumption that the nonspecific **esterase** is a chymotrypsin-like enzyme.
IT Mast cells
 (esterases in, inhibitor effect on)
IT Cinnamic acid, hydroxy-
 Phosphoric acid, diethyl p-nitrophenyl ester
 Sodium arsenite
 (effect on adenosinetriphosphatase, **esterase** and leucine aminopeptidase of mast cells)
IT Et₄P₂O₇
 (effect on adenosinetriphosphatase, esters and leucine aminopeptidase of mast cells)
IT Nicotinamide, adenosinetriphosphatase
 (**esterase** and leucine aminopeptidase in mast cells in response to)
IT Sodium fluoride, adenosinetriphosphatase
 (**esterase** and leucine aminopeptidase of mast cells in presence of)
IT Benzoic acid, p-(chloromercuri)-, adenosinetriphosphatase
 Lead nitrate, adenosinetriphosphatase
 Magnesium chloride, adenosinetriphosphatase
 Physostigmine, adenosinetriphosphatase
 Silver nitrate, adenosinetriphosphatase
 Taurocholic acid, sodium salt, adenosinetriphosphatase
 (**esterase** and leucine aminopeptidase of mast cells in relation to)
IT Sodium azide, adenosinetriphosphatase
 (**esterase** and leucine aminopeptidase of most cells in relation to)

IT Acetic acid, phenyl-, adenosinetriphosphatase
(**esterase** and leucine aminopeptidase of most cells response
to)

IT 1,2,3-Indantrione, monohydrate (Ninhydrin), adenosinetriphosphatase
(esters and leucine aminopeptidase of mast cells in relation to)

IT Esterases
(in mast cells, inhibitor effect on)

IT Leucine aminopeptidase, L-Leucine aminoexopeptidase or Leucyl peptidase
(of mast cells)

IT 50-00-0, Formaldehyde 7447-39-4, Copper chloride, CuCl₂ 7487-94-7,
Mercury chloride, HgCl₂ 10108-64-2, Cadmium chloride
(adenosinetriphosphatase, **esterase** and leucine aminopeptidase
of mast cells in relation to)

IT 7646-79-9, Cobalt chloride, CoCl₂
(adenosinetriphosphatase, **esterase** and leucine aminopeptidase
of mast cells in response to)

IT 64-69-7, Acetic acid, iodo-
(adenosinetriphosphatase, **esterase** and leucine aminopeptidase
of most cells in relation to)

IT 51-28-5, Phenol, 2,4-dinitro- 55-91-4, Isopropyl phosphorofluoridate,
(C₃H₇O)₂FPO 60-00-4, Acetic acid, (ethylenedinitrilo)tetra- 70-34-8,
Benzene, 1-fluoro-2,4-dinitro- 103-71-9, Isocyanic acid, phenyl ester
108-24-7, Acetic anhydride 143-33-9, Sodium cyanide 321-54-0,
Coumarin, 3-chloro-7-hydroxy-4-methyl-, diethyl phosphate 371-86-8,
Phosphorodiamidic fluoride, N,N'-diisopropyl- 546-71-4, Phosphonic acid,
ethyl-, ethyl p-nitrophenyl ester 7631-89-2, Sodium arsenate
7718-54-9, Nickel chloride, NiCl₂ 7757-83-7, Sodium sulfite, Na₂SO₃
(effect on adenosinetriphosphatase, **esterase** and leucine
aminopeptidase of mast cells)

IT 100-56-1, Mercury, chlorophenyl- 128-53-0, Maleimide, N-ethyl-
27323-35-9, Benzoic acid, iodoso-
(effect on adenosinetriphosphatase, esters and leucine aminopeptidase
of mast cells)

IT 7773-01-5, Manganese chloride, MnCl₂
(mast cell adenosinetriphosphatase **esterase** and lucine
aminopeptidase in relation to)

IT 7646-85-7, Zinc chloride
(mast cell enzyme response to)

IT 9000-83-3, Adenosinetriphosphatase
(of mast cells)

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